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CLINICAL ASPECTS OF EPILEPSY.[1]

By JOHN F. WILLIAMS, M.D., B.S., (Melbourne), M.R.C.P. (London), D.P.M. (London),

Honorary Psychiatrist, Saint Vincent's Hospital and Children's Hospital, Melbourne.

IT gives me great pleasure and I feel it a great honour to open this discussion on a subject of fascinating interest and of undoubted importance.

The problems of epilepsy have aroused interest and discussion from the days of Hippocrates at least, as is shown by his treatise "On the Sacred Disease" in which he protests against the prevalent belief that it was to be regarded as a visitation of a god or demon and states that it "has a natural cause from which it originates like other affections."

[1] Read at a meeting of the Section of Neurology and Psychiatry of the Victorian Branch of the British Medical Association on March 31, 1930.

In spite of this we find that until recent times the epileptic has been regarded as possessed and treatment has been directed to driving out the evil spirit with varying degrees of success.

With the rapid growth of neurology during the latter half of the last century, epilepsy became a centre for much research and experiment, such men as Hughlings Jackson, Mercier, Bastian and Ferrier having contributed to our knowledge. Still more recently numerous contributors to this problem in all its aspects have appeared and what is to follow is derived from many sources with a special indebtedness to the work of Muskens and Kinnier Wilson.

Phenomena.

Epileptic seizures may be said to be characterized by two manifestations, a disturbance of consciousness and a motor activity. These may occur

together, but each may occur alone, so that the manifestations of the epileptic process present the widest variations.

The best known examples are, of course, the attacks of *grand mal* and *petit mal*, but there are other attacks which seem to be closely allied, and these have recently been attracting attention as a result of the emphasis by Kinnier Wilson on their clinical variety but probable identity from the standpoint of physiological process.

In the first place we have the difficulty of observation of the attacks themselves and the necessity for relying on the statement of the patient and his friends and relatives, and this difficulty can only be overcome by observation in hospital, often for prolonged periods, when the patient is in apparently good health. This is generally impossible under present circumstances.

With reference to *grand mal*, it is obvious that the classical type is often not adhered to, and numerous variations occur in different individuals and at different stages of the disease. It seems that prolonged duration of the attacks tends to make each attack conform more to the classical type, but it is noticeable that in early cases variations are comparatively common. *Petit mal* also presents many clinical syndromes, but if a patient has both major and minor attacks, it is generally the case that the same aura, if any, occurs in each.

Jacksonian, cortical or epileptiform attacks are of very great importance, owing to the fact that they may afford a clue to the localization of a pathological process in the cortex which may be removable, but it is important to note that the commonest cause of Jacksonian attacks is idiopathic epilepsy and that Jacksonian attacks may lead to major attacks.

The existence of sensory Jacksonian attacks with a characteristic sensory sequence, sometimes associated with a paralysis apart from any convulsion, is also to be noted.

The so-called "variants" may be briefly reviewed, for, though some are relatively uncommon as isolated attacks, they are nevertheless of great clinical and physiological interest, and some may occur as aura to an attack of *grand mal*.

Myoclonic epilepsy will receive attention later. *Epilepsia partialis continua* is rare and consists of a twitching of one segment of the body which is practically continuous between the convulsions. Tonic attacks without clonic movement are also rather rare and are sometimes, though not always, associated with a definite lesion, generally cerebellar. Coordinated epilepsy refers to the fact that the movements at times resemble voluntary movements, though often aimlessly repeated; they may occur before, during or after the convulsion or may be the only motor phenomena in *petit mal*.

Under inhibitory epilepsy we find cases in which immobility seems to be epileptic in origin, and under this heading it seems advisable to include the attacks of narcolepsy, that is, paroxysmal sleep, which Adie would place together as a separate disease. Still more does it seem necessary to include the cases of cataplexy, that is, paralysis and tonelessness often brought on by emotion, under this heading.

Reflex epilepsy is generally applied to cases in which there is a definite sensory excitation of the attack, for example, flicking the nose or loud and unexpected sounds or injuries to peripheral nerves, and these will be referred to later. It has also been applied to those cases supposedly due to other irritations, such as phimosis or worms.

Sensory epilepsy has already been mentioned, but its similarity to the complicaed aura which occur in uncinate fits, sometimes in association with localized tempero-sphenoidal disease, may be pointed out, and also the possibility that certain cases of trigeminal neuralgia may be epileptic in origin.

Affective epilepsy will also be referred to later as following stimuli with an emotional significance, but it is advisable here to point out that a loud and unexpected sound generally causes an unpleasant affect which may be that of fear, so that the differentiation of reflex and affective epilepsy may be difficult.

"Psychical variants" or equivalents have been applied to the moods of irritability *et cetera* that may precede a fit and also to some of the post-paroxysmal fugues, automatisms *et cetera*, but in some cases with an epileptic inheritance, the only apparent abnormality may be repeated attacks of uncontrollable bad temper. Visceral variants are very undefined and the possibility of epileptogenous foci occurring in sympathetic centres may be mentioned. Gowers used the term vaso-vagal attacks and it seems probable that some syndromes referable to the cardiac, respiratory, digestive, urinary or vasomotor systems which occur periodically, may be epileptic, with the focus possibly in the dorsal vagal nucleus and the vasomotor centre. Other attacks such as excessive sweating or yawning or fears of impending harm have also been ascribed to epilepsy.

It will be obvious that these manifestations cover a wide field and seem to grade into conditions such as tics and syncopal attacks which are not regarded as epileptic; but it seems that no useful purpose is fulfilled by limiting our conception of epilepsy to attacks of *grand* and *petit mal* when these allied attacks may occur sometimes in the same patient from a similar physiological process.

Reaction Types: Not a Disease.

The difficulties arising in an attempt at definition of epilepsy are increased by the fact that similar attacks to the above occur in many clinical conditions. These may be roughly classified as arising in patients with endogenous toxins and metabolic dyscrasias, such as diabetes, hypoglycæmia, uræmia, rickets and eclampsia; with exogenous toxins, such as alcohol, lead, bismuth, syphilis; with acute infections, as in the pyrexial convulsions of children, or malaria; with organic cerebral states, such as

tumour, trauma, hæmorrhage, thrombosis, meningitis or encephalitis; with cardio-vascular conditions, such as arteriosclerosis and heart block, or with various psychoses, such as *dementia præcox*.

In cases of so-called idiopathic or essential epilepsy we can find no such ætiology, though attempts have been and are still being made to prove the existence of some such factor as cerebral gliosis or alkalosis in these also.

In view of this state of affairs it seems regrettable that we should still cling to the idea of epilepsy as a definite disease entity and should instead regard it as a reaction type which may be brought about by a variety of influences. As has been so often stressed of late, a disease is merely a convenient concept under which we group a number of cases of ill health because of certain common characteristics which may be ætiological, biochemical or biophysical, psychological, pathological or merely descriptive. These characteristics may at times be more or less united and we may have a series of cases showing to some extent common ætiological factors, physiological and psychological reactions, pathological findings and a uniform course, such as cases of lobar pneumonia. In no case can our knowledge be said to be complete, but it should be the aim of medical science to make the chain of events in a disease process as full as possible. This is not helped by the elevation of syndromes such as epileptic attacks into diseases where none of the above factors can be said to be known.

Physiological Basis and Experiment.

If we put all cases which show attacks similar to those in "idiopathic epilepsy" together, we find that a common feature seems to be a discharge of energy which differs from the normal output in leading to phenomena which are sudden, excessive and temporary, though these may not always be invariable characteristics. This seems to be due to the release of certain mechanisms from inhibition.

As a result of much clinical and physiological work, Muskens has recently attempted to enlarge the sphere of myoclonic epilepsy so that it includes at least 50% of ordinary epilepsy, and this work seems to be of some importance in connexion with this release of mechanisms.

He has found that in cats which have been given suitable doses of camphor monobromide, there occurs a condition closely resembling that in many cases of human epilepsy and that by varying this dosage a graduated series of phenomena may occur.

At first there is noticed an increased tendency to show myoclonic reflexes. These are activated by tactile or acoustic stimuli which are unexpected and of suitable intensity. They have been studied in many untreated animals, including man, and consist in a rapid clonic contraction followed by a slower tonic contraction which may or may not lead to movement of a body segment. They are akin to other reflexes and are characterized by a prolonged refractory period, a short latent period and a marked tendency to after-discharge. Later in the same animal there may occur myoclonic shocks which are similar movements, but in this case the stimuli are unknown, though it may be that the tendency to the reaction in these cases is greater and that a less powerful, unrecognized stimulus will bring about its release.

These two sets of phenomena are to be found very markedly in some human epileptics (myoclonic or regional epilepsy) and various figures are given as to their incidence. Reynolds stated that they occur in 81% of epileptics, while Muskens, from careful examinations of the histories and observation of his patients, gives the percentage in two thousand epileptics as being males 55 and females 65. They are liable to be missed in case taking, unless attention is directed towards them, as they are often overlooked by the patient and his relatives, and for this reason other authorities have apparently underestimated their frequency.

Associated with these shocks there may be slight dulling of consciousness and articles may be dropped. They are especially frequent in the early morning or on retiring to bed and are often described as the "jumps."

It is of interest that they also occur in apparently normal people. Children are particularly prone to sudden starts on tactile or acoustic stimuli and to night starts accompanied at times by terror, while the sudden jump just before or after falling asleep is a very common condition in adults, especially when fatigued. Charles Darwin was struck by the activity of these reflexes in the natives of South America when exposed to positions of danger.

It is found that if the dose of the drug is sufficiently large, animals suffer from series of these shocks and eventually react by a convulsion which is very similar to the major attack in man; for there seems to be a prodromal period of malaise with ruffled fur, irritability *et cetera*; then follow definite tonic and clonic stages and later a period of exhaustion and confusion with apparently automatic behaviour in which the animals (especially monkeys) may seem to be hallucinated. Shortly after this the animal recovers and appears to be rid of the toxic substance, though in other cases another attack follows which then succeeds in detoxication, if we are to judge from the general condition of the animal and from the fact that no shocks occur and no reflexes can be elicited by stimuli which were formerly adequate.

Other attacks have been noticed which in some ways resemble the *petit mal* in the human being, but these do not seem to possess the same power of detoxication. He has also shown that an impending attack can be prevented or compensated for by violent muscular exertion or the eliciting of repeated myoclonic shocks, and these activities are therefore thought to have a similar detoxicating effect. This seems to apply in some cases of human epilepsy where the attack may be worked off by violent exertion.

The tonic stage is shown to be fundamentally the same as the clonic stage, though in the former the

rate of movements is much more rapid, and this is in accord with the views of Hughlings Jackson and most modern authorities, though others have thought that the tonic stage is a function of the basal ganglia while the clonic stage is due to cortical recovery from a generalized inhibition.

In claiming that these attacks fulfil a definite physiological function in ridding the animals of the drug, he draws a comparison with other phenomena such as inflammation, pyrexia and rigors, which are essentially protective, but may become pathological.

The transition from the stage of myoclonic shocks to the convulsion is ascribed to an alteration in the refractory period which permits an "after-discharge" such as is described by Sherrington in reflex responses to stimuli of a certain intensity, in the scratch reflex, for example, where the after-discharge takes the form of similar repeated movements.

The main centre for these responses is thought, as the result of numerous sections of tracts, to be ponto-medullary, though the influence of the pyramidal and other cortico-fugal tracts on this centre is admittedly very great.

It seems that this work in tracing the evolution of the epileptic convulsion from myoclonic reflexes and showing that they seem to fulfil a useful function merits careful consideration as giving an approach to the physiological processes at work in epileptics.

If we assume that there are reflexes such as these, we are still faced with the problem as to what leads to their being activated.

Causation.

In the causation of the great majority of disorders it is impossible to affix one isolated cause, though the importance of causative factors vary in any one case; and in epilepsy it seems that there are various factors with the power to fire off this mechanism if we are to judge from the fact that the convulsions occur in the course of many disorders apart from those in which the ætiology is still largely unknown. It seems, therefore, that not only from the descriptive standpoint and the different areas which are the origin of the discharge, but also from the standpoint of ætiology we would be wiser to speak of the epilepsies than of epilepsy.

It is necessary to realize that any view of the organism which entails a separation from the environment, is artificial and apt to be misleading, for it is obvious that we live owing to our relations with the environment, and that our reactions are largely conditioned by stimuli, that is, by changes in the environment with which we come into contact. It is also obvious that the environment includes not only the external medium, but also the internal medium in which we live. To take an example, the oxygen of the air certainly does not cease to be a factor in the environment when it reaches the lungs nor yet when it reaches the blood stream, for here it is still part of the environment of the body tissues.

Certain of the relations between the organism and the environment take place mainly at metabolic levels, others at reflex levels, visceral and somatic, others again at higher levels where they are called instincts, habits or conditioned reflexes, while higher still are the levels where deliberation and conscious thought play a prominent part. It is, however, necessary to realize that these levels are not separable and that any attempt to differentiate them by calling in mind or consciousness is futile, for metabolic reactions affect our conscious thought and thought affects our metabolism, while conscious activities readily become automatic.

A disease represents faulty reaction of the complete organism and not of one individual organ, though this may appear predominantly affected in the end results as seen in autopsy.

In all reactions of the organism the response depends not alone on the stimulus, but on the significance which this has acquired for the individual during its racial or personal past. We find, therefore, that certain essential responses at the metabolic and reflex levels are very deeply ingrained in the organism and relatively invariable, while others less essential may be markedly influenced by passing events, as is shown, for example, by Pavlov's work on conditioned reflexes and Watson's work showing that the knee jerk, for example, may be activated by a stimulus of light in a specially trained animal. In the case of epilepsy it seems, therefore, that we should consider the changes in environment or stimuli which bring about the attacks, that is, the exciting causes, the significance which may attach to these by virtue of the individual organization (the predisposing causes including the epileptic personality) and the responses which occur. The latter have already been touched upon with a hypothesis regarding their physiological nature.

The differentiation between the predisposing and exciting causes is often difficult, but certain factors may be briefly considered which seem to be of clinical interest.

The occurrence of a fit is often heralded by various prodromal symptoms. These are emphasized by Muskens as signs of intoxication or charge, for example, such phenomena as coated tongue, flatulence, anorexia, dilated and unequal pupils, prolonged headaches, vasomotor disturbances, fainting fits, feelings of oppression and irritability, while there are also other symptoms pointing to partial discharges, such as myoclonic shocks and attacks of *petit mal*. These may serve to warn an observer than an attack is imminent and at times measures may be taken to prevent the convulsion or guard the patient from harm.

It is impossible to differentiate these symptoms clearly from the various aura which immediately precede the attack in about 30% to 50% of the cases, and it is thought by Muskens that these too are signs of intoxication. Following these we have the convulsion which serves to throw the toxin into the blood stream, and as a result there is a period

of post-paroxysmal phenomena before the toxin is finally rendered innocuous.

It is possible that in a percentage of cases this interpretation of the attack as due to a toxæmia, possibly alimentary in origin, or to a metabolic dyscrasia does hold; and other experimental work, such as that on the insulin hypoglycæmic convulsions which bring about an increased blood sugar, gives it further probability.

It must, however, be recognized that in many cases no source of this toxin or other proof of its existence has been found, and it seems advisable to cast round for other possible factors which may activate these mechanisms.

Assuming, as we probably should, that every individual is liable to react by convulsions under certain conditions, we find that some are predisposed by inheritance or other disease.

The rôle of inheritance in epileptics has evoked discussion, but there seems to be no doubt that many epileptics are heavily burdened in this respect. Muskens gives the figures as being approximately 24% showing direct inheritance, and Russell Brain gives the percentage in which there was an epileptic family history as being 28% in epileptics, as opposed to 10% in ordinary hospital patients. Kinnier Wilson and Pierre Marie, for example, tend to minimize this factor somewhat, the former pointing out that even these figures leave 70% to 80% in whom there is no definite history of an epileptic inheritance. Pierce Clark states that 20% have a normal inheritance.

Other predisposing factors are insanity, alcoholism or syphilis, though how these tend to epilepsy is unknown.

In all cases we have to realize that there is a definite tendency for epilepsy to become a habit, so that once there has been a convulsion from any cause, there is a greater danger of a fit recurring even under other conditions; or in the words of Gowers: "Epilepsy is self perpetuating."

Patients with a deficiency of mental development are especially prone to epilepsy and this is probably to be correlated with a diminished power of inhibition of the process, though other factors, such as faulty metabolism, may also play a part.

The effect of organic cerebral disease, such as trauma, tumour *et cetera*, may be ascribed to faulty inhibition, associated perhaps with excessive irritability of these mechanisms. It is, however, of interest to note that it is in quite a small percentage of cases of cerebral trauma, 5% to 10%, that epilepsy develops, so that we may also have to assume a predisposition in these cases. The epileptic habit may develop in these patients and any attempt at surgical relief must be followed by careful medical treatment. The rôle of birth trauma is doubtless important, but investigation is difficult and statistics vary widely.

Another group of cases are those in which there is a definite history of febrile illness in childhood associated with signs of a cerebral lesion, pointing to an encephalitis, and these seemed to make up 13% of Musken's cases, while a history of infantile convulsions, apart from any indication of encephalitis, occurred in another 11%, so that it seems probable that infantile convulsions, especially if due to encephalitis, predispose towards epilepsy. Patrick and Levy give the increased risk as 5 to 1.

This may be variously interpreted as rendering the epileptic mechanism more susceptible either by definite cerebral lesions or merely by a process of facilitation as in the epileptic habit. On the other hand, it may speak for an inherent defect of metabolism, such as a tendency towards alkalosis which carries with it a predisposition towards epilepsy.

Another factor which has been brought forward, is intolerance to certain proteins, and cases of cessation of attacks on omission of certain articles of food are quoted.

The question of there being a definite personality which predisposes towards epilepsy, has been discussed for many years, but has recently been brought into prominence by Pierce Clark in America. Doubts have been freely expressed as to the marked predominance of such characteristics as excessive supersensitiveness, egocentricity with lack of consideration for others, emotional poverty and liability to emotional outbursts if thwarted, rigidity of ideation and mentation and general lack of adaptation to the social environment which he describes as characteristic and showing a predisposition, and other authorities, such as Aldren Turner, stress a general emotional attitude of anxiety and fear.

The question as to whether these personality trends occur before the fits and act as a predisposing cause, or after the fits as a result of cortical deterioration, or of the isolation and feeling of inferiority and insecurity which they produce, or whether the two go hand in hand as a result of some other process, cannot be definitely settled; and it is probable that each of the above may be true for a certain percentage.

Another factor in causation to which attention should be directed, is that of emotional stress and the effect of the social environment. This is a point on which much controversy has taken place. Gowers gave emotion, such as fright, excitement or anxiety, as the most potent exciting cause of epilepsy, and this was, of course, long before the days of psychoanalytical interpretations of the fit as symbolizing a return to the mother's womb away from the stress of life. Since that time its importance has been classed at various times as almost negligible and of supreme importance.

This divergence of opinion is probably due to the different preconceived ideas of investigators and the neglect of a dynamic view of the organism which has led to the study of disordered structures, rather than that of morbid relations between the organism and the environment.

There does seem to be a good case for the investigation of many epileptic seizures as allied to con-

ditioned reflexes or chain reflexes; and this view may to some extent be correlated with the work of Muskens, already discussed, for here too the attack may be regarded as protective.

The type of case which renders this approach possible, became more marked during the war, and the terms reflex, affective or psychogenic epilepsy have been applied.

Numerous cases have been quoted in which attacks in no way differing from the classical epileptic convulsions have appeared as a response to stimuli having a profound emotional significance, for example, fear, for the individual.

Such cases as these so impressed Rows and Bond that they published a discussion of epilepsy as a "functional" disease and quoted cases of cure as a result of investigation of the emotional life and reeducation. Their work has been regarded by some as irrelevant and dealing with hysteria, but the cases quoted do not seem to bear this out.

Treatment.

If sufficient care with case taking is observed, cases occur which seem to warrant an investigation along these lines, and it seems that this type of case is far more common than we have been led to believe in the past; but whatever views we may hold regarding their ætiology, there is no doubt that an important factor in the treatment of all epilepsies is the placing of the patient where he is freed from harmful stimuli. This point is being increasingly stressed by modern authorities who emphasize the need for careful investigation of the patient's environment and the alteration of this, if possible, so as to avoid emotional stress, dietary indiscretions, over-fatigue and so forth.

The patient may by these means be freed from fits, the epileptic habit be broken and later a return be made to a simple mode of life without the necessity for the prolonged, intensive, sedative treatment which is still so common and often essential in our present state of knowledge.

I feel that I have trespassed unduly on your time and patience, but the epilepsies are a fascinating and important problem and one which is well ripe for investigation from all aspects. It has been quite impossible to touch on more than a few of these, but others will be dealt with by succeeding speakers. To me it seems, however, that the first approach to this problem should be from the clinical side, with intimate investigation of the onset of the malady with any possible significance attaching to the situations in which this occurred, and an endeavour to avoid similar situations or to rid them of their significance and harmful effect.

This may seem to be a Utopian dream in many cases, but it seems to have a firm basis in physiological and psychological research and gives a hint of a line of treatment more rational than those at present adopted, with the possible exception of the recent advent of the ketogenic diet which does apparently rob the epileptic response of its stimulus or the stimulus of its significance for the organism in about 30%. The necessity for early and thorough treatment should not need to be stressed. The aim of diagnosis should not be the placing of a patient in a convenient diagnostic pigeon hole with a routine treatment attached, but the understanding of his condition, and it is with this aim in view that the problem of the epilepsies should be approached.

THE PATHOLOGY OF EPILEPSY.[1]

By Reg. S. Ellery, M.D. (Melbourne),
Neurologist to the Victorian Lunacy Department; Clinical Assistant to the Psychiatrist, Melbourne Hospital.

An apology is needed for the title of this paper. The pathology of epilepsy is unknown, and that which passes for such is largely inadequate and unsubstantiated. Most of it is blushingly tentative, some of it frankly fantastic. In fact the whole field of speculative causation is scattered with moribund theories and embryonic guesses, ranging from demoniacal possession to the "local liberation of histogenic convulsant poisons," and such a plethora of observed gross pathological concomitants as not infrequently has tended to cloud the neural genesis of convulsions.

Kinnier Wilson(1) has said that "there are many organic nervous states of which epileptic fits are a symptom; in fact the epileptic fit is nothing else but a symptom." This may be perfectly true. But while at first sight it would seem that he has relegated the classical fit and its variants to the secondary realm of symptoms, one may yet argue and even believe that one of the many organic nervous states of which the epileptic fit is a symptom is epilepsy itself. This possibility has led to confusion, and the introduction of the words "idiopathic" and "essential" as qualifying adjectives to epilepsy—as an entity and not a syndrome—has become a species of clinical camouflage, making confusion worse confounded.

As a result clinicians have recently broadened their conceptions and speak rather of "the epilepsies," and the pathologist has adopted a similar caution, preferring to speak of the "convulsive state" rather than of the epileptic fit. All this has come about in the last decade or so following a very widespread and concentrated endeavour to trace the pathological process which must lie behind the polymorphic phenomena of the epilepsies. For it does not matter whether you subscribe to the belief, hallowed by custom, that the epilepsies are diseases of the brain or whether you agree with Oppenheim that they are "toxicopathic conditions with cerebral manifestations," pathogenetic obscurity still confronts you.

The manifestations of epilepsy—using the term in its widest connotation—are manifestations of a disordered neuronic mechanism. While there is no

[1] Read at a meeting of the Section of Neurology and Psychiatry of the Victorian Branch of the British Medical Association on March 31, 1930.

constant anatomical lesion in the epilepsies and only a minority of patients with extensive cerebral pathological change have fits, yet there is always some lesion found within the central nervous system of epileptics, although it may only be discovered at autopsy.

In a series of two hundred and fifty-nine *post mortem* examinations on patients presenting epileptic manifestations, the late Dr. W. A. T. Lind[2] found the following:

Thickening of the dura	99
Dura thickened and adherent to calvarium	36
Dura adherent to calvarium but not thickened	14
Pia thickened	127
Pia thickened and adherent	20
Sclerosis of the *cornu ammonis*	37
Hypergliosis of brain	63
Ventricles dilated	134
Ependyma of lateral ventricles granular	4
Ependyma of lateral recess of fourth ventricle granular	16
Ependyma of *calamus scriptorius* granular	18
Abnormalities of brain pattern	80
Hydrocephalus ex vacuo	20
Plaques jaunes	11
Tumour of the brain	9
Cerebral softenings	17
Pachymeningitis hæmorrhagica	6
Atheroma of vessels of the brain	23
Macroscopical changes in chorioid plexus	7
Trephine operations	7
Flattened convolutions	8
Heterotopia	2
Internal hydrocephalus	1
True porencephaly	1
False porencephaly	1
Asymmetry of hemispheres	15
Gross wasting of gyri	71
Old lesions of the brain substance not determined	7

The time honoured sclerosis of the *cornu ammonis* which Lind found in only 14% of his cases, but which other observers in the past have regarded as the classical lesion, the minute areas of softening, chronic gliosis and the proliferation of glial fibres in the external layers of the cortex, are not now accepted as pathognomonic of the convulsive state, but are more correctly regarded as degenerative, having been observed as part of the histopathology of the non-epileptic heredo-degenerations. And there is no doubt also that many among the gross necropsy findings of Lind must be classified as degenerations and developmental abnormalities, as the result of or accompanying the convulsive phenomena, rather than causal agents.

Gross pathology and cerebral histopathology have not so far been fruitful in the elucidation of the cause of epileptic convulsions, and having explored this field and found it seemingly sterile, many observers have sought to determine whether there were abnormal humoral and vascular influences at work on the cerebral cortex, the manifestations of which were being exteriorized in the kinetic discharge of the convolution. An enormous amount of work has lately been performed in the realm of humoral chemistry and on the neurology of vascular mechanisms. It will be quite impossible even to refer to the greater part of this work, suggestive as some of it undoubtedly is. But one may say generally that, like the cerebral pathology, while many abnormalities have been found in humoral pathology, there is yet no constant finding pathognomonic of epilepsy.

For example, the relation between food and fits has been recognized since the time of Hippocrates, and some clinicians, following Collier, believe that the whole secret of the convulsive state lies in a metabolic dyscrazia, but the biochemistry of metabolism in epileptics gives no constant abnormality in support of this. Pathological increase in blood fibrinogen and spinal fluid protein has been advanced as evidence of abnormal protein metabolism. Results relating to fat metabolism are at present somewhat conflicting, although it is established that many patients show a reduction of seizures when placed on a high fat diet. But the effect of this ketogenic diet, as it is called, is to cause alteration of the acid-base equilibrium which would appear to be the *modus operandi* of its therapeutic action. The carbohydrate metabolism appears to be but little altered in epileptics. Dr. Farran-Ridge who worked on this subject in England some years ago, tells me that the majority of his epileptic patients gave blood sugar curves within normal limits. For a number of years the liver, on account of its detoxifying power, has been subject to suspicion in the pathology of epilepsy. Autopsy studies have shown that in the majority of chronic epileptics the normal ratio of the weights of brain and liver is reversed, the liver weighing less than the brain.[3] It has also been established that the majority of epileptics show a liver dysfunction, as shown by their abnormal response to the lævulose tolerance test. Other evidence has been adduced to support this liver dysfunction, but its nature remains quite obscure. Abnormalities of the gastro-intestinal tract seem to be no more common in epileptics than in those suffering from other nervous diseases.

The cerebro-spinal fluid gives no constant abnormality, some patients showing high pressure readings without abnormality of the chemical constituents, while in others the total protein content is above the normal, and in others again, depending on the albumin-globulin ratio, colloidal gold curves of luetic and meningitic types may be observed.

Recent work has shown that it would be difficult to overestimate the importance of a detailed knowledge of the hydrodynamics of the spinal fluid circulation in the pathogenesis of the majority of intracranial conditions, and this applies no less to the pathology of the convulsive state, as has been recently demonstrated by Temple Fay.[4]

It is now generally accepted that the cerebro-spinal fluid is a dializate from the chorioid plexus, the latter acting as a semipermeable membrane setting up a Donnan equilibrium between the blood plasma and the cerebro-spinal fluid. In health, therefore, the chorioid plexus maintains an electrolytic equilibrium between plasma and fluid, the rate of production being dependent on the intracranial

blood pressure. From the experiments of Weed and McKibben and of Dandy and Blackfan we know that the cerebro-spinal fluid has a definite circulation, passing from the lateral ventricles by the foramina of Monro into the third ventricle and from here along the Sylvian aqueduct to the fourth ventricle. It leaves the fourth ventricle through the foramen of Magendi and the lateral foramina of Luschka to pass into the spinal subarachnoid space and the large basal cisternæ, over the convexity of the cerebral hemispheres to the Pacchionian bodies situated in the superior longitudinal sinus. Fay,[4] from histopathological studies, considers that the Pacchionian bodies, being the chief means of egress of the cerebro-spinal fluid, play a most important part in the production of supracortical œdema which appears to predispose to the convulsive state. The Pacchionian bodies, evolved from a small collection of cells associated with the large venus sinuses and known as the subarachnoid villi, are said to reach their adult state about the twentieth year of life.

Fay writes:[4]

> They are dependent upon the laws of development and growth similar to any other body tissue. They are probably prone to the same degree of variability as other anatomical structures . . . . It is reasonable to suppose that they are open to the same degree of congenital malformation or abnormality that might be expected elsewhere. That hereditary deficiencies in this structure may explain their absence or failure to develop is conceivable. That inflammatory or toxic conditions in the mother during pregnancy may affect them is possible. That birth injury, hæmorrhage or subsequent trauma due to situation at the vertex, open fontanelle and movement of the cranial bones in the early months of life is probably the most common cause of their damage. Inflammation, either direct or secondary to infection, toxæmia, encephalitis, meningitis, septic or aseptic, trauma, direct or with adjacent or distant subarachnoid hæmorrhage, tumour or eclampsia affect these structures and may so impair their function that they may at once, or as a chronic increased deficiency occurs with advancing age and growth of the brain associated with increased demand, reach a point of failure to compensate, thus permitting the accumulation of excessive amounts of subarachnoid fluid over the cortex.

That all this discussion is not merely of academic interest is borne out by the application of therapeutic procedure, for by limiting the fluid intake which tends to promote cerebral dehydration, a great reduction in the number of an epileptic's seizures may be effected, just as the condition of *status epilepticus* may be alleviated by spinal drainage through lumbar puncture.

The work on the mechanism of supracortical œdema, brought forth by Fay, dovetails in with that of Lennox and Cobb[5] on the variation of the acid base equilibrium in epilepsy. Put shortly, these investigators claim to have demonstrated that acidosis tends to inhibit and alkalosis to augment epileptic seizures. A condition of acidosis, they claim, may be brought about by: (i) Fasting; (ii) ketogenic diet, that is, a diet rich in fats and poor in carbohydrates; (iii) the ingestion of acids or acid-forming salts; (iv) vigorous physical exercise; (v) temporary rebreathing.

"Procedures which will induce seizures in susceptible individuals will not do so if the inhaled air contains an increasing amount of carbon dioxide."[6] And likewise hyperpnœa or forced breathing by promoting a rise in blood alkalinity causes seizures in the susceptible.

The question is, when is a patient "susceptible"? And seeing that œdema gives rise to decreased oxygen tension in the tissues, the answer may well be that the susceptible person is one in whom there exists as the result of plastic arachnoiditis, malformation, disease or sclerosis of the Pacchionian bodies a supracortical œdema. For the researches referred to point to the fact that "increased permeability of the capillaries, increased intracranial pressure and œdema are three phases of the same process," or, put aphoristically, alkalosis is associated with tissue œdema, acidosis with dehydration.

It is not suggested that the foregoing is in any way assumed to be a fully-fledged explanation of the mechanism of the epileptic fit. It is but a modest presentation of some of the modern work which is being executed in the now hopeful field of neurophysiology. While it would be both impossible and absurd to underrate the value of a sound anatomical basis to neurology, it would seem that this subject has been too long dominated by the anatomist. For there comes a time when the study of structure is sterile unless it can be coordinated with that of function. Neurological advance in the future will surely depend upon the extension of a functional pathology, and in no case can this be more true than in the pathology of epilepsy.

That which is here presented is merely an extension of the teaching of Hippocrates who said of epilepsy that "whosoever is acquainted with such a change in men and can render them humid and dry by regimen could also cure this disease." But for nearly two thousand years the rational outlook of Hippocrates was replaced by the paralysing concepts of religion. Epilepsy became the "sacred disease." Pathology was founded on the Pentateuch. Disease was dominated by demons. Even now the obscurity of its causation cannot be denied; but in the bright sunrise of science these "mechanical" theories stand as finger-posts pointing the way to solution.

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WHEN WILL AUSTRALIA ADOPT MODERN PROPHYLACTIC MEASURES AGAINST LEPROSY?

By Sir Leonard Rogers, M.D., F.R.C.P., F.R.C.S., F.R.S., I.M.S.Ret.

The leprosy problem in Australia is a comparatively small one, yet Dr. C. Cook's three years' investigation showed that it is far from being solved. Thus in 1924 he pointed out that, in spite of leprosy having been a notifiable and quarantinable disease for three decades, it is still as prevalent as ever in some of the States, and in his 1927 report he showed that the disease in Queensland, after declining between 1910 to 1919 from 85 to 42, steadily increased again from 1919 to 1927 from 42 to 77, especially among the Europeans. He thought that leprosy was under control in New South Wales, but this is not altogether borne out by recent data, for in the seven years up to 1920 the total segregated cases varied between 20 and 24 and the yearly admissions averaged three; and from 1920 to 1927 the admissions numbered 26, an average of 3·25, and the decline in the total remaining to 17 at the end of 1927 was due to five repatriations and 15 deaths during that period.

The Failure of Rigid Compulsory Segregation to Stamp Out Leprosy.

Only about one decade ago we had nothing better to offer poor lepers than compulsory imprisonment for many years or for life, although in no other chronic disease is such a penalty enforced. Three years' intensive study of leprosy literature of the previous six decades convinced me that rigid compulsory segregation has never yet succeeded in stamping out leprosy and rarely even in reducing it materially in any warm climate, such as Hawaii, the Philippines, the West Indies or even in South Africa, although a humane method, with no compulsion except in the case of indigents for the first three decades, and only a very modified form later, has reduced leprosy to small proportions after seventy years' efforts in Norway. As the evidence regarding segregation is given in detail in "Leprosy" I need not labour the point here, as it is now generally admitted.

Reasons for the Failure of Rigid Compulsory Segregation.

Nor are the reasons far to seek, for even under very favourable European conditions in Norway only one leper in five was discovered and isolated within the first three years after the appearance of the first symptoms of the disease; this accounts for it having taken three-quarters of a century to reach the present position in Norway with no new cases for several years past. Again, in the great Culion settlement of the Philippines the average duration of the disease on admission was eight years, and although advanced cases are now rarely seen in the towns, yet the flow of new patients, infected from others during the eight years before they were isolated, shows little diminution after a quarter of a century of rigid compulsory segregation. In South Africa Dr. Mitchell, a strong supporter of compulsory measures, wrote in his 1927 report:

> The discouraging fact, however, is that nearly all the new admissions are advanced cases of four, six or more years' duration, some of them already past the more infectious stage, and most of them unpromising for successful treatment.

He also admits that early cases are hidden, and I can vouch for the fact that even patients with uninfective leprosy have come all the way from South Africa to England for treatment because they dared not go to a doctor in South Africa for fear of isolation with those in advanced stages of the disease. Only last month Dr. Victor G. Heiser, the distinguished author of the great Culion isolation experiment, candidly told me that it had largely failed because the supply of new patients was kept up indefinitely through the infection from lepers hidden for years before being isolated for fear of that measure, and it is very significant that the Philippine authorities, with a unique experience of the modern treatment of leprosy, have now modified their policy to allow those in comparatively favourable stages to be treated at special hospitals near large towns without being sent to Culion, and, still more, that they are now treating patients in the early stages at "skin clinics" without any isolation, as extensively done for a number of years in India following my first trial of this plan in Calcutta, commencing in 1915.

The Clinical Curability of Early Leprosy and its Bearing on Modern Prophylaxis.

It is now over a decade since I demonstrated that many of the patients in the comparatively early stages of leprosy met with among the out-patients of a large Calcutta hospital could be cleared of all clinical signs of active leprosy and rendered bacteriologically normal and uninfective by weekly injections of soluble preparations of the active parts of chaulmoogra and hydnocarpus oils, but that the advanced lepers met with in asylums are much less amenable to treatment. A summary of subsequent evidence in confirmation of this discovery will be found in my recent Cameron Prize Lecture before the Edinburgh University, so I need only mention a very few data regarding the curability of early cases. Among 486 Hawaii patients treated in the course of five years only 8% of advanced infections were cleared up, but 38% of moderately advanced ones and no less than 64% of patients in the early stages recovered clinically. In the Culion settlement only 15% to 20% of the advanced infections there seen cleared up under prolonged treatment, but a much larger proportion of earlier ones in the special hospitals did so, and the total recoveries now amount to over 2,000 cases, a number which speaks for itself and requires no amplification.

The Proportion of Early to Advanced Cases Found by Leprosy Surveys.

In areas with compulsory segregation the true number of lepers is quite unknown on account of the early cases being hidden for fear of the measure.

The Indian Branch of the British Empire Leprosy Relief Association, however, have solved this problem by means of surveys organized by Dr. E. Muir, and they have established the fact that for every one advanced, easily recognized, census-returned leper there are four or five early unrecognized cases. Is it any wonder, then, that compulsory segregation of the advanced patients fails to reduce the disease materially, and are not those who fondly imagine they can stamp out leprosy within a reasonable time by isolating the one-fifth of advanced sufferers, after they have had ample opportunities for several years in which to infect others, living in a fool's paradise? It follows that in direct proportion to the liability of compulsion to lead the early curable patients to be hidden until they have reached a relatively incurable and more infectious condition, compulsion at the present time will do more harm than good.

The Mediaeval View that Leprosy is Highly Contagious is Untrue.

The data I collected on the contagiousness of leprosy are given in Table VII on page 78 of "Leprosy," from which it appears that only from 2% to 5% of persons living in the same house with a leper contract the disease, and conjugal infections occur in a similar proportion of exposures. The disease is thus probably less infectious than tuberculosis.

The Conditions under which Infections Occur and the Incubation Period.

In the same work, in Table IX, page 83, I showed that about 80% of infections were due to living in the same house with a leper, while in about 30% of the 700 cases I collected it followed sleeping in the same bed with a leper, while the nodular cases are twenty times as infective as nerve cases, so the latter are commonly harmless; yet in some countries they form the majority of isolated patients. In South Africa, however, Dr. Mitchell adopted my suggestion to release all patients with bacteriologically negative "burnt out" nerve infections, which amounted to one-third of the whole, with great saving of expense. I have also shown that the incubation period is less than five years in 80% of cases, while among children in the Philippines it averaged only three and a half years.

The Modern Prophylaxis by Curative Treatment of Early Leprosy Cases Detected by Frequent Examinations of Contacts.

For some years past I have advocated the following simple method of prophylaxis based on the foregoing facts. The households and other close contacts of all known cases of leprosy and of each newly discovered case should be examined from head to foot for the earliest signs of the disease, and this examination should be repeated at least every six months for a period of five years, by which means some 80% of possible infections from each should be discovered in the early stages and the great majority of them cleared up before they have reached a contagious stage or infected any others, with the consequent elimination of most of the sources of infection. By repeating this process for another five years very few foci of infection would remain and the disease would be under control.

Under the Above Method Compulsory Segregation Would Become Obsolete in About Two Decades.

If the infectious patients were also isolated, the above plan would render segregation unnecessary in about twenty years or less for the simple reason that the duration of life of the patient with the highly infectious nodular form of the disease is only eight to ten years, while the mixed and nerve infections would have lost any infectivity by that time, so the problem would be solved or reduced to very small proportions.

The Value of this Plan has been Proved in the Case of Nauru Island.

The difficulty was to get this method tested under favourable conditions, but fortunately I was consulted some years ago by Dr. Dew about the serious outbreak of leprosy in the mandated island of Nauru and wrote advising its trial there. Earlier in this year Dr. G. Bray reported the results in a paper he read before the tropical diseases section of the Royal Society of Medicine, in which he showed that although about one-third of the population were infected with leprosy, the isolation of the bacteriologically positive patients on one side of the island and the treatment of "negative" patients as out-patients without isolation, together with monthly examinations of all the healthy people of the small population for new infections, has proved so successful that the number of remaining cases has been reduced by about one-third in three years. Still more important, not a single very early treated case has gone on to the highly infectious nodular stage, so that the problem of dealing with an outbreak of leprosy, wherever this plan is feasible, may be considered to have been solved, and leprosy should be reduced to very small proportions at Nauru after a few more years. The contrast between the rapidly successful results at Nauru with the failure of three decades of compulsory segregation in Queensland is too evident to require further emphasis. The only question that remains is the modifications of the old compulsory segregation system which are necessary to enable the modern method of finding and treating all lepers in the earliest stages of their disease to be used.

The Success of Voluntary as Opposed to Compulsory Isolation in Tropical Africa.

The number of lepers in British tropical African colonies is now estimated at not less than 150,000, while any attempt at compulsory segregation of them only leads to their being hidden. Yet 4,000 are now under treatment in Nigeria alone, and many thousands in our other African colonies and territories, under a system of providing colonies with

land to cultivate on a purely voluntary basis, all within about four years, and about one million doses of "Alepol" (sodium hydnocarpate) were supplied to Africa alone last year by the British Empire Leprosy Relief Association.

The Modification of Rigid Compulsory Segregation to Allow Early Diagnosis and Treatment.

In parts of tropical Africa where compulsion is not in operation, I am opposed to introducing it in any form which is likely to cause patients in the early stages to be hidden, but in countries already possessing a costly segregation system, including Australia, I have always advised that it should not be abolished at once, but only modified so as to insure that patients in the early stages come forward for treatment instead of hiding themselves until they are far less amenable to it and have had time to infect others. For this purpose the following plan has been adopted in Mauritius and is about to be legalized in British Guiana at my suggestion. A board of at least two medical officers, including a pathologist, shall examine all persons with suspected leprosy and if on bacteriological examination Hansen's bacillus is not found, as happens in nearly all early nerve and most very early skin cases, they should be treated as out-patients at the nearest medical institution, or privately if well-to-do, but inspected every three months for infectivity. Infective persons may still be isolated, but with skilled treatment, without which I consider the measure unjustifiable, and it will be remembered that Dr. Cook reported in 1927 that the treatment in some of the Australian leper asylums was not very efficient, in spite of the New South Wales cases costing £200 per segregated patient. Doubtless that has since been rectified. If this measure is combined with frequent examination of all contacts for early symptoms, as advised by Dr. Cook in his 1927 report, then I feel sure that Australia can look forward to being nearly rid of the scourge of leprosy in two decades or so. One advantage of retaining this modified compulsion, for a time at least, is that pressure can be exercised on patients in the early stages to attend regularly for treatment under penalty of being segregated if they fail to do so. As charge of a leper asylum gives little or no experience of treatment in very early cases, I also advise that every medical man, before taking charge of such an institution or of other leprosy work, should attend a course of instruction in Dr. E. Muir's leprosy clinic at the Calcutta School of Tropical Medicine, such as are given several times a year, including, I think, in November.

It is for the individual States to decide how far they can carry out the new methods of prophylaxis, but I am convinced that the nearer they can get to the ideal plan, which is proving so successful at Nauru, the more rapidly will they succeed in solving their problem with regard to the rapid eradication of leprosy from their country.

CYCLIC VOMITING.[1]

By Idris Morgan, M.B., Ch.M. (Sydney),
Honorary Physician, Newcastle Hospital, New South Wales.

Definition.

Attacks of vomiting which are recurrent, which afflict children below the age of puberty, and which are always accompanied by the excretion of acetone in the urine and expired air, are described as cyclic vomiting. It can be stated almost dogmatically that recurrent vomiting in which acetone cannot be detected in the urine and/or the breath during an attack, is not due to the faulty metabolism believed to be the cause of true cyclic vomiting.

Ætiology.

Age.

The first attack may be encountered at any age before the tenth year. Rachford[(1)] reported a case in a nursling two months of age. It is rarely seen in breast-fed infants. I saw three attacks of cyclic vomiting in a child between the second and third birthdays.

Diathesis.

Children who suffer from these attacks of cyclic vomiting, possess the diathesis to which the term "arthritic" is sometimes applied. On investigation it will be found that one or more members of the family have suffered or still do suffer from migraine, diabetes, asthma, arthritis, urticaria *et cetera*. Many of the sufferers themselves alternate attacks of asthma and urticaria with their attacks of vomiting. The French school classes these as "equivalents" of cyclic vomiting. One of my patients suffered from very frequent widespread urticaria. The treatment to be described later caused the attacks of cyclic vomiting to cease five years ago and with them the urticaria.

Another child suffered from frequent attacks of asthma. He did not vomit. The removal of infected tonsils and adenoid tissue did not reduce the number or severity of his attacks. The treatment used for cyclic vomiting was recommended and caused a cessation of the attacks of asthma. At my request the mother discontinued it. The asthma returned, to disappear on resumption of treatment.

T. G. Oliphant[(2)] reported twenty cases and stated that "no case of severe vomiting with ketosis occurred in any but fair children." I have seen three sufferers who are brunettes and one of whom became semi-comatose in an attack which lasted seventy-two hours.

Climate.

Umana, practising 2,600 feet above sea level, reported a large number of cases and it would appear that in high altitudes the attacks are more

severe. The mortality in Umana's series of cases was higher than in any other series reported to that date (1926).

Exciting Causes.

The inherent faulty metabolism which is believed to predispose to attacks of cyclic vomiting, will be dealt with in another section. Certain exciting causes are well recognized. The list given here is culled from various authors and from observation of the small groups of patients who have come under my care; it is as follows:

1. Fatigue and excitement. Children who suffer from cyclic vomiting seem to become fatigued easily. The children in whom I have observed this syndrome, are usually bright, intelligent and "always on the go." The excitement of dressing for a fancy dress party seemed to precipitate an attack in one patient. The parents asserted that undue excitement of a similar character had had a similar result previously.

2. Indulgence in foods containing a high percentage of fat, particularly animal fat, notably cream, egg yolk, chocolate and ice cream, is the exciting cause which has been most recognized.

3. The onset of an acute infection.

4. Eruption of teeth.

5. Constipation. Marfan sounds a note of warning against the use of calomel which he says in some cases has been observed to precipitate an attack.

There is the usual group, of course, in which no exciting cause can be inculpated. The much used "idiopathic" is hardly applicable. In my cases many lapses from the narrow path of dietetic righteousness to the broad highway of chocolates and ice cream at birthday parties have resulted in urgent calls to relieve the youthful sinner's vomiting.

The Attack.

It is not proposed to describe the attack in detail, as all practitioners have seen it frequently, and as it has been described so often in other articles. It must be emphasized, however, that a definite diagnosis of cyclic vomiting cannot be made unless acetone is detected in the urine and breath at some stage of the attack and unless the child is under the age of puberty. This is a very important point, as will be seen when the question of differential diagnosis is discussed.

The first vomiting may be followed by a period of quiescence, sometimes of several hours' duration. Acetone can be smelt in the breath, however, lassitude is always apparent and the vomiting recommences. Water may be retained for ten minutes or half an hour, but is then returned and there is a repugnance to all food. The attack ceases suddenly and the child frequently asks for food soon after it has ceased.

Prognosis.

Death during an attack is a very rare event. Torres Umana reported several cases, mostly in children under six years of age. In each case "coffee ground" vomit was observed. The patients became comatose. The respiration became deep, sighing and irregular and ceased a few hours after the onset of full coma. Fatal results have been observed mostly in very young or very delicate children. The prognosis is almost always good. The attack ceases suddenly and the recovery is as rapid as the onset. The appetite returns almost at once and a demand for food may be regarded as a certain sign that that particular attack is over.

Diagnosis.

The history of the child and its family is important, particularly when the medical attendant is called to treat a first attack. However sceptical one may be concerning the classification of the *genus homo* into groups of diatheses, it will be found that children suffering from true cyclic vomiting always belong to a family conforming to the type described under the heading diathesis above.

In regard to the presence of acetone in the urine and breath, sometimes the smell of acetone precedes the onset of vomiting. Usually it is detected with ease if the child is asked to exhale. It is invariably present in the urine and in most cases at the onset of the vomiting. It is wise, if no acetone is detected in one specimen and an attack of cyclic vomiting is suspected, to examine the urine at frequent intervals, as the amount of acetone excreted varies from time to time during the attack and also does not vary directly with the severity of the vomiting. The best test is that of Legal (glacial acetic acid, sodium nitroprusside and ammonia), both because it is the easiest to perform and because the depth of the coloured ring is a moderately accurate guide to the amount of acetone present in the specimen. Usually the acetone disappears from the urine in a few days. The smell of acetone may be detected in the breath for as long as forty-eight hours after the vomiting has ceased. Marfan records occasional cases in which acetone has been detected in the urine for many days after the attack.

Diacetic acid is almost always present, but β-oxybutyric acid is not often found. Sugar is never present. The urinary chlorides are diminished during the attack and are eliminated in excess after it. As would be expected from the loss of weight and malnutrition, the total nitrogen of the urine is increased. This malnutrition and rapid emaciation give the patient a characteristic appearance termed by the French *maigre*. No single English word gives the exact shade of meaning.

Persistent vomiting, drowsiness and a loathing for food occur. In many cases it may be noticed that immediately after vomiting the child is temporarily brighter and says that he or she feels better.

The onset is sudden and in the majority of cases occurs at night.

Differential Diagnosis.

If the above points are kept in mind, particularly the pathognomonic sign—acetone in the urine or breath—a diagnosis can always be made. The physician must, however, bear in mind that an attack of cyclic vomiting in persons of this diathesis is equivalent to the convulsion or rigor which sometimes marks the onset of an acute illness. It is common to see any of the exanthemata and/or acute attacks of upper respiratory tract infection ushered in by an attack of cyclic vomiting.

I have seen two cases of appendicitis in children accompanied by cyclic vomiting of sufficient interest to warrant a brief record.

Case I: A male, aged six years, had a history, obtained from the parents, of very frequent "bilious attacks." The patient was vomiting almost continuously. The temperature was 40° C. (104° F.), the pulse rate 120. (Many attacks of cyclic vomiting are afebrile.) The urine contained acetone. The abdomen was soft, but the child complained of abdominal pain. Pressure over the right iliac fossa elicited tenderness which was not very pronounced. A radiogram, taken a few days later, revealed a long appendix well filled with bismuth. The appendix at operation was found to contain pus. After the operation the attacks of cyclic vomiting were less frequent. Attacks of asthma from which the patient also suffered, disappeared altogether.

Case II: A female, aged eleven years, had a family history that revealed migraine in the grandmother, an uncle on the father's side and in the father. A brother suffered from cyclic vomiting. The patient had had six or seven attacks of cyclic vomiting, none of them severe. Occasional flatulent dyspepsia for three or four months preceded a typical severe attack of cyclic vomiting. A specimen of urine passed one hour after the onset of the vomiting was heavily laden with acetone. The temperature was 36·9° C. (98·4° F.) and the pulse rate 120. The presence of pain referred to the epigastric and umbilical areas and tenderness on pressure over McBurney's point led to a diagnosis of acute appendicitis. The vomiting commenced at 5 a.m. and continued till 12 noon, when, under ether anæsthesia, an appendix containing much fluid pus was removed. The temperature at 11 a.m. was still 36·9° C. The anæsthetic did not seem to intensify the ketosis.

It seems that in both these cases an inflamed appendix acted as an exciting cause of attacks of cyclic vomiting.

Pathology.

The urine is always highly acid. Howlett and Marriott showed that the alkaline reserve of the blood is less than normal during an attack. Ross and Josephs in one case found a definite diminution of the blood sugar and they state that always hypoglycæmia will be found if the blood be examined at the height of an attack of cyclic vomiting. Later P. S. Cammidge[3] described two hundred patients suffering from chronic hypoglycæmia (fasting blood sugar of 70 milligrammes *per centum* or less). He suggests that a "fundamental defect in the liver" is the cause and stresses the hereditary factor. Cyclic vomiting was noted in thirty-two patients under the age of nine years. His table shows that all persons under nine years of age in this group of two hundred hypoglycæmics seen by him suffered from cyclic vomiting and further a family history of vomiting was obtained in 46% of those who were beyond the age when cyclic vomiting occurs. Prior to Cammidge's observations the hypoglycæmia during the attack was attributed by some to inanition. A number of records of *post mortem* examination of subjects dead of coma incident to an attack of cyclic vomiting, reveal a constant finding, namely, fatty degeneration of the liver and kidneys. In this connexion it is interesting to note that the acidosis produced by chloroform causes the same fatty degeneration of the liver and kidneys and that the breath smells not of chloroform, but of acetone.

Theory Concerning the Cause of Cyclic Vomiting.

Glucose is required for the oxidation of fats in the body. Schaffer[4] showed that one molecule of glucose oxidized is accompanied by the oxidation of two molecules of fatty acid in the body. The oxidation of fats proceeds only as far as the stage at which ketone bodies are formed, unless glucose is also being oxidized by the internal secretion of the pancreas. It is well known that, as in *diabetes mellitus*, when the body is unable to metabolize glucose, the oxidation of fats is incomplete and ketone bodies appear in the blood and urine, causing the recognized symptoms of the disease.

In cyclic vomiting there is no such loss of power on the part of the body to metabolize glucose. On the contrary the liver can take up glucose and convert it into glycogen and the attack can be aborted or abbreviated by giving sugar. The only successful treatment of the attack of cyclic vomiting is based on this very fact. It may be appropriate here to mention that this observation also distinctly contraindicates the use of "Insulin" in cyclic vomiting, because there is nothing wrong with the pancreas which can supply all the insulin needed. "Insulin" is used to oxidize sugar which the body cannot metabolize. In cyclic vomiting there is no impairment of the glucose oxidation function. If the sugar can be got into the intestine, the liver will and does take it up, metabolize it and so restore the balance and cause the vomiting and acetonæmia to disappear. It has been established that there is a condition of hypoglycæmia present during an attack. Cammidge's observations suggest that it is very probable that this hypoglycæmia exists in a less marked degree throughout life in individuals of this diathesis. There is, therefore, either a permanent low glycogen store in the liver or a store which is more rapidly depleted when called upon than is the case in other individuals. As soon as this glycogen reserve is used up, the oxidation of fats is incomplete and ketosis occurs. Clinical evidence supports this theory strongly. Excess of animal fats in the diet is observed with striking constancy as an exciting cause of cyclic vomiting.

A certain amount of glucose is required to enable the oxidation of these fats to be carried out completely. Fats are oxidized completely only in the presence of glucose, which is being oxidized itself by "Insulin." The subject of cyclic vomiting has

not a sufficient reserve of sugar. What he has is oxidized and thereafter the oxidation of fats must proceed in the absence of glucose undergoing oxidation. Ketone bodies are thus formed, enter the blood stream and cause the attack of vomiting. At the onset of an acute infection, during excessive exertion or excitement, the carbohydrate store is called upon to provide extra energy. Carbohydrates are more easily and quickly burned and converted into energy than fats and proteins. The glycogen store is depleted, there is no glucose to be metabolized, fat metabolism stops short and again the clinical picture of ketosis presents itself. Cammidge says:

> A congenitally low blood sugar figure usually causes no serious inconvenience so long as the patient's defective metabolism is not subject to any extraordinary strain, but severe physical or mental exertion rapidly causes collapse, while ketosis, sickness and other symptoms due to an insufficient supply of available carbohydrate develop from causes which would have little or no effect upon a normal individual.

Cyclic vomiting is frequently ascribed to "fat indigestion." This appears to me to be an inaccurate term. Too much fat is stored which has to be oxidized to provide even the energy required for the maintenance of life during sleep after the low carbohydrate reserve is used up. This explains why the attack of cyclic vomiting so frequently commences at night. The carbohydrates taken at the evening meal and all the liver reserve are used up during the hours which follow the last intake of food. Then the body calls on fats for energy and these are oxidized in the absence of the oxidation of glucose and ketosis occurs as has been explained above. It is not fat indigestion. The trouble comes after the fat has been digested and stored in the tissues. Undoubtedly the ketosis is ultimately produced as a result of incompletely oxidized fats, but these children can and do digest fats quite well, provided there is enough sugar present to undergo the oxidation which is the necessary accompaniment for the complete digestion of fats. Fats cannot be completely oxidized unless sugar is being oxidized at the same time. When a number of patients with cyclic vomiting were collected in a hospital in London for observation, their diet was not interfered with, but none of them suffered from cyclic vomiting. It was discovered that the sister in charge of the ward was giving them a daily ration of malt. The extra sugar enabled them to deal with the fat in the diet quite well. Sugar deficiency seems to be at the root of the matter. These children are born with a hereditary inability to maintain a sufficient reserve of sugar. (Compare Cammidge's findings quoted above.) When this poor reserve is depleted, diacetic acid enters the blood stream and is regarded as the chemical compound which causes most of the symptoms seen in acidosis. It is converted into acetone in the lungs and bladder.

Treatment.

Treatment of the Attack.

Withhold all food. Give glucose and water continuously. Five or six teaspoons of ordinary cane sugar dissolved in a glass of water to which 0·6 gramme (ten grains) of sodium bicarbonate has been added, is a satisfactory method. It should be given continuously, even though vomiting is frequent. Usually the fluid is retained for from twenty minutes to half an hour and some of it passes on to the duodenum and is absorbed. Apparently a small quantity of sugar in the liver is sufficient to prevent further formation of ketone bodies. I have frequently aborted an attack in one patient by giving the above mixture, and have seen so many attacks reduced in duration by the same method of treatment that there is little doubt of its efficacy. The administration of glucose prior to an anæsthetic to prevent post-anæsthetic acidosis has become a routine procedure.

This procedure is of special value when the operation of subtotal thyreoidectomy is performed. When thyreotoxicosis is present, the liver glycogen is rapidly mobilized and the advantage of insuring a good reserve beforehand is obvious. Also in operations upon the gall bladder, when an infection from the biliary duct system is suspected of having involved the pancreas and depressed its function, the same procedure is valuable as a safeguard. It is in these latter cases that "Insulin" may be used with advantage in combination with the exhibition of sugar. "Insulin" is contraindicated in an attack of cyclic vomiting, so also is calomel. Purgatives by mouth are usually vomited. In severe cases glucose *per rectum* is indicated. In spite of statements to the contrary, glucose is absorbed from the large bowel, but not from the stomach. Sixty to one hundred and twenty cubic centimetres (two to four ounces) of a 10% solution, depending upon the age of the patient, repeated in six hours, may be given.

Investigation shows that there is a definite diminution of the alkaline reserve and therefore it seems reasonable to give alkalis in the form of sodium bicarbonate. This substance, however, has no effect on the symptoms, as is to be expected. It neutralizes some acid, but quantities sufficient to act as a purgative (a not uncommon action of large doses of sodium bicarbonate) failed to render the urine alkaline or diminish the vomiting and drowsiness in one case, and not till glucose was given did these symptoms improve.

Sodium bicarbonate plays no part in the chemical reactions necessary for the oxidation of fats and therefore cannot be expected to correct a syndrome due to a breakdown of this reaction. The same result is obtained, as far as relief of symptoms is concerned, if the sodium bicarbonate is left out of the mixture described above. The sweet taste of the sugar solution, to which some of the small patients object, may be modified by the addition of lemon juice.

Treatment Calculated to Prevent Attacks.

Eliminate excess of all foods containing animal fats from the diet, chiefly cream (skim the milk), chocolate, egg yolk, ice cream and brains. There is

no necessity to exclude all these articles completely. They are all rich in proteins, vitamins and fats and are among the most valuable all round articles of diet. It will be found that each child can deal with a certain amount of animal fat. It will also be found that by adding a measured quantity of extra glucose to the diet every day the child can deal with an increased amount of fat. Some parents soon learn to superintend the diet in such a way that the child is not deprived unnecessarily of these valuable foodstuffs, but at the same time does not receive sufficient to cause ketosis.

Increase the consumption of glucose. Two or three lumps of loaf sugar after or between each meal have been found sufficient in my experience. The extra sugar has not been found to spoil the appetite or the teeth, or upset the bowel, or do any of those dire things of which we have heard so much. Why should it? These children want sugar and use it to keep up a store which becomes too rapidly depleted.

Marfan recommends courses of small doses of bicarbonate of soda in doses of 0·12 to 0·3 gramme (two to five grains) taken with sugar twenty to thirty minutes before meals on a certain number of days each month. He states that alkalis taken fasting and in small doses "stimulate metabolism and actuate organic combustion." He reports benefit in many cases from courses of alkaline mineral waters such as Vichy, Vals *et cetera*. He also advocates the use of "alkalis in slightly larger doses than usual" with sugar during an attack. He says that they do not act only as simple neutralizers of acid, but stimulate the metabolic function of the liver. There is no doubt that the record of his success in treating and aborting attacks of cyclic vomiting shows that at any rate alkalis, used in the doses mentioned, do not do much harm. I have found that regulation of the diet alone will prevent attacks completely and certainly without the use of alkalis at all. I have aborted attacks, sometimes prior to the actual onset of vomiting and sometimes soon after the vomiting has commenced, by the use of sugar and alkali and have also produced the same results with sugar alone.

It is interesting to compare Maclean's views on sodium bicarbonate in this connexion with those given above, especially in view of the time honoured custom of giving alkalis freely in ketosis.

Maclean says that it is wrong to give alkalis in ketosis, because they tend to reduce sugar tolerance and prevent the storage of glycogen. It has been found that if sodium bicarbonate is given with fifty grammes of sugar, often sugar is found in the urine, though the blood sugar curve does not rise. A coworker in his laboratory found ketone bodies in his urine after starving for forty hours. He then took fifty grammes of sugar and found that the ketone bodies disappeared from his urine in from one to one and a quarter hours. He repeated this experiment twice a week for some months. He found further that if he took sodium bicarbonate with the glucose, his urine still contained ketone bodies nine hours afterwards. This was the longest period for which he could endure the starvation. Maclean therefore concludes that it is not correct to give sodium bicarbonate to children suffering from ketosis and states that they should be given sugar alone. He also holds that alkalis interfere with the action of "Insulin."

In comparing these two statements it must be borne in mind that Marfan gives small doses (0·3 to 0·6 gramme or five to ten grains) of bicarbonate of soda with relatively large doses of glucose and it is probable that under such circumstances the action of the alkali in delaying the disappearance of the acetonæmia is not so strong. In practice the use of sodium bicarbonate does not seem to be necessary.

Use of Glucose in the Treatment of Allied Conditions.

It is too much to expect that the prescription of a daily extra ration of sugar will prove a panacea for all those ills to which those possessing the arthritic diathesis are heir; nevertheless, the use of glucose in this manner, as will be seen from the discussion above, is based on sound principles and does provide some pleasant surprises.

The table given by Cammidge in his article on chronic hypoglycæmia is most illuminating, and the recently reported success in certain cases of asthma following the use of sugar and alkalis seems to confirm his findings. It is interesting to note that the patient with asthma, mentioned in the earlier part of this article, lost his attacks when he swallowed extra sugar without alkalis. The results of an extra daily ration of sugar in three cases of migraine treated within the last few months are interesting as an illustration of the possibilities of this form of treatment in some cases of this troublesome complaint.

Case III: F., aged thirty-two years, had a family history of migraine. She had suffered for many years from migraine. Attacks were very severe, causing complete incapacity for from twenty-four to forty-eight hours; they were also very frequent, seven to ten per month. The patient was advised to reduce the intake of animal fats to a minimum and a daily ration of two to three lumps of loaf sugar after each meal was prescribed. The frequency and severity of the attacks were reduced immediately. Prior to the commencement of the treatment, the patient had suffered from a severe attack every three or four days for over two months. After the institution of treatment there were two much less severe attacks only in four weeks. It is four months since treatment was commenced and the improvement has been maintained. There have been no further attacks.

Case IV: M., aged forty-six years, a railway official, was seen on June 14, 1930, in a typical attack of severe migraine. He gave a history of frequent severe headaches. He stated that they were becoming more frequent and more severe. The increase in severity coincided with his promotion in the service to a position of greater responsibility. Later he detailed a long list of treatments he had tried without success. It was difficult to know what advice to give. Foci of infection had long since been eradicated. Half-heartedly he was advised to leave drugs alone and take three lumps of sugar after each meal. No advice in regard to elimination of fat was given. He looked decidedly astonished and did not come back to

report progress as requested. Five weeks later his wife came specially to report a great improvement. Perhaps it was the sugar, perhaps it was the cessation of aspirin, phenacetin and caffein citrate, but it seems probable that perhaps he suffered from chronic hypoglycæmia. An attempt is being made to find out.

CASE V: M., aged thirty-eight years, a brother of the patient described in Case III, was at one time incapacitated for several years by frequent severe attacks of migraine. The attacks were now occasional. When about to leave his office to catch a train to Sydney, he experienced flashes before the eyes which he knew to be the precursor of an attack of migraine. Within a short time he was in the throes of a severe attack. He drank a glass of water in which ten lumps of loaf sugar had been dissolved. The attack was relieved and he caught a train three hours later. He reports that the headache disappeared completely and that he felt quite well.

This man had suffered hundreds of attacks and had never been able to abort an attack before. He is not susceptible to suggestion. He possesses a medical degree and therefore, let us say, his statements are reliable. The train journey, as would be expected, caused a slight return of the symptoms which were checked by taking more carbohydrates.

Acknowledgement.

In conclusion I should like to acknowledge my indebtedness to Dr. F. W. D. Collier for his collaboration in the section "Theory Concerning the Cause of Cyclic Vomiting" of this paper and also for supplying me with notes on Maclean's views on the use of bicarbonate of soda in ketosis.

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(1) A. B. Marfan: *"Les Vomissements Périodiques avec Acetonémie."*
(2) T. G. Oliphant: "Acidosis in Childhood," THE MEDICAL JOURNAL OF AUSTRALIA, August 17, 1929, page 216.
(3) P. J. Cammidge: "Chronic Hypoglycæmia," *The British Medical Journal*, May 3, 1930, page 818.
(4) H. E. Roaf: "Text Book of Physiology."

Reports of Cases.

MULTIPLE PATHOLOGICAL LESIONS.

By D. B. ROSENTHAL, M.D., B.S. (Melbourne),
Resident Medical Officer, Caulfield Convalescent Hospital.

MULTIPLICITY of lesions, as discovered at autopsy, is not an unusual finding; nevertheless, the case described displays a disparity of multiple lesions such as merits publication.

M.J., a female, aged seventy-three, died at Caulfield Convalescent Hospital following a history of dyspnœa, palpitation and the usual symptoms attendant to cardiac failure. At autopsy the following lesions were discovered in the organs specified:

1. Severe degree of aortitis, with marked atheromatous changes affecting the aorta from the commencement at the arch to the division into the common iliac arteries. There were numerous plaques of deposited lime salts with the formation at parts of atheromatous abscesses. The coronary orifices and particularly the orifices leading to the intercostal arteries were considerably reduced in size. The heart itself showed a marked degree of myocarditis, with a large, thick-walled left ventricle; the coronary vessels showed changes similar to those in the aorta, but no definite occlusion was detected. There was marked degree of chronic inflammatory change in the mitral and aortic valves, with adherence of cusps of the latter, and calcification had occurred in both, being most marked at the attachments of the cusps of the mitral valve to the heart wall.

2. There was a basal pneumonic condition of the right lung which, together with an engorged and œdematous state of the left lung, were terminal conditions. In addition, the right lung was small and generally contracted by chronic fibrosis, with a large pleural effusion, occupying the basal half of the anterior part and the diaphramatic part of the right pleural cavity. This cavity was lined with organizing lymph and the whole appearance suggested an inflammatory rather than a transudatory process.

3. The gall bladder was small and contracted and contained about eighty to ninety small faceted gall stones; the wall of the gall bladder was of "strawberry" type. In addition, there was a single gall stone in the cystic duct.

4. The right kidney was a pyonephrotic sac, with merely a shell of renal tissue left; firmly fixed in the renal pelvis was a single calculus, roughly spherical, and about 1·25 centimetres (half an inch) in diameter.

5. The liver was the subject of marked fatty change, with an obvious cirrhotic element; there was patchy perihepatitis.

In addition to these departures from the normal, there was present an umbilical hernia through a fat abdominal wall; general anasarca was noted.

VISCEROPTOSIS ACCOMPANIED BY MELANCHOLIA.

By T. J. BIGGS, M.B., Ch.M. (Sydney),
Willoughby.

MRS. B., aged thirty-eight, married, with one child of fourteen years, came under observation on May 6, 1930. Her complaints were long and many and she had been in impaired health for ten years, during which time she had been treated for "a complete nervous breakdown" on three occasions. For three years past she had suffered with "very bad nerves," frontal headache, anorexia, constipation and more particularly with insomnia, incessant fatigue and dragging pains from the axillæ down the chest sides, loins and thighs. The menstrual history varied little from normal until three months ago, since when the periods had been scanty and of one day duration only.

On examination the patient was thin (about 44·1 kilograms or seven stone in weight), very emotional, tremulous and most illogical on interrogation. The abdomen was of the long, narrow type with a narrow subcostal angle; the kidneys were easily palpable and there was generalized tenderness on pressure below the umbilical level. Apart from exaggerated reflexes, no other deviation from the normal was detected as physical signs.

The patient was submitted to X ray examination by Dr. H. M. Cutler who reported marked evidence of gastro-enteroptosis with some reflex duodenal irritability, also marked coloptosis, with hepatic and renal ptosis.

The stomach was shown very low in position, with the pylorus and duodenal cap below the iliac crest level, dilated, sagging, freely mobile and hypotonic. The duodenal cap showed evidence of irritability, but no organic lesion.

The coils of small bowel were shown lying for the most part well below the iliac crest level, with the appendix poorly filled and retrocaecal.

The colon was poorly filled and definite ptosis of the colon was present, the hepatic and renal flexures showing 3·75 centimetres (one and a half inches) above the iliac crest level.

A skiagram showed both kidneys low in position, the right shadow showing the lower border at the iliac crest level; the shadow of the liver margin was low.

The patient was then treated along Weir-Mitchell lines and during the ensuing three weeks there was alleviation of the insomnia, anorexia, constipation and pains.

On May 28 she complained of severe hæmorrhoidal pain, and other local measures failing, relief was afforded by local injections of quinine and urea hydrochloride.

On June 22 she developed a most severe septic throat with fever of 39·4° C. (103° F.) beginning as an acute follicular tonsillitis, then spreading with a white exudate round the soft palate to the uvula and pharyngeal walls. A streptococcus was recovered on culture from the tonsils.

Up to this stage there had been no obvious manifestation of mental impairment. The fever, however, was accompanied by hallucinations, both visual and auditory, and these persisted after the subsiding of the throat infection. To these hallucinations was added a phase of acute melancholia, the vivid experiences of the nocturnal terrors being recounted by the patient in the morning to the accompaniment of much weeping.

On June 28 Dr. W. R. Page visited the patient in consultation and was of the opinion that the mental state was associated in part at least with the apparent premature menopausal changes; he suggested "Hormotone" *plus* sulphonal and "Somnos" as adjuvants in treatment.

On July 2 the clinical picture changed from that of acute melancholia and hallucinations to that of profound stupor, in which the patient lay listlessly in bed with the eyes closed, mouth open, tongue dry and furred, resisting food and unresponsive to any but strong stimuli. Forced feeding with the spoon was resorted to. She remained in this state for four days, after which she improved rapidly, the mental confusion becoming less acute and taking food more easily.

On July 9 she insisted on getting up from her bed to a lounge and for the next five days was in a better state than at any time of her illness; nevertheless, though her speech for the most part was logical, she was still easily mentally confused.

On July 14 the patient was transferred from her home to a private hospital, and within one day of the change of environment she again lapsed into a state of profound stupor, with moderate fever, in which she remained till her death nine days later, on July 25. This state was interspersed with short periods of comparative mental activity attended by acute melancholia.

Incontinence of urine and fæces, muscular wasting and signs of congestion of the lung bases were manifest six days before the end.

On July 18 Dr. Page again visited the patient in consultation; examination did not reveal any evidence of an organic lesion of the nervous system.

The interesting points of the case are:

1. The visceroptosis had evidently remained unrecognized over a period of years. What difference (if any) would have resulted from the early adoption of a suitable abdominal belt?
2. The onset of the menopause was apparently premature. Was this primary or secondary to or even unassociated with the mental derangement.
3. There were phases of profound stupor resembling semi-coma alternating with acute melancholia accompanied by hallucinations.
4. Profound psychoneurosis complicated the condition.

A BROKEN PNEUMOTHORAX NEEDLE.

By George M. Hay, M.B. (Sydney),
Honorary Physician, Mater Misericordiæ Hospital, North Sydney.

The experience about to be described is probably not by any means unique, but it seems to give ground for speculation as to whether some of the types of needle at present used in the operation of inducing artificial pneumothorax are constructed with the utmost possible regard for the safety of the patient.

A male patient, aged thirty-eight, was found to be suffering from unilateral pulmonary tuberculosis, X ray examination disclosing a small cavity in the left upper lobe. As there were no apparent contraindications and it was impossible from an economic point of view for the man to relinquish his occupation in order to undergo sanatorium treatment, induction of artificial pneumothorax was recommended and the advice accepted.

A preliminary puncture was made in the fifth intercostal space, in the mid-axillary line, but, owing to the fact that adhesion between the visceral and parietal pleura appeared to be present in this situation, a second attempt to enter the pleural cavity was made in the seventh space in the posterior axillary line.

A Rivière needle, with annular grooves marking off the cannula into divisions each one centimetre long, was used. As soon as it was considered that the needle had commenced to enter the intercostal space, the sharp-pointed trocar was withdrawn from the cannula as a preliminary to inserting the blunt one used for puncturing the parietal layer of pleura on the occasion of the first air introduction.

An instant after the withdrawal a slight cracking sensation was experienced by myself. Concluding that the cannula had broken, I attempted to introduce the blunt trocar through it in order to locate the broken-off piece, but was unsuccessful.

I then cut down, under "Novocain" and adrenalin local anæsthesia, in an attempt to pick up the broken-off piece, working along the proximal portion of cannula which I had left *in situ*. This procedure was also unsuccessful, as the proximal portion had apparently swung to some extent out of position and I was unable to realign it.

Having abandoned the attempt to extract the broken needle immediately, I communicated with Dr. Alan F. Oxenham, Honorary Radiologist to the Mater Misericordiæ General Hospital, who kindly made an X ray examination of the patient at that institution and located the fragment as being at the level of the seventh thoracic vertebral spine 11·25 centimetres (four and a half inches) from the mid-line.

With this as a guide I made a fresh incision, again under "Novocain" and adrenalin infiltration anæsthesia, over the area where the foreign body had been localized.

Due mainly to the accurate work of Dr. Oxenham, I was able, by entering the interval between the *teres major* and *latissimus dorsi* muscles, to extract the fragment from where it lay, embedded in the intercostal muscles and just above the eighth rib. The broken-off fragment was three centimetres long and had snapped through one of the annular grooves which is a feature of this type of needle.

This experience would appear to show that a cannula graduated in this manner is sometimes unsafe. When I afterwards examined the one which was concerned in the accident just described, I found that the thickness of its wall at the site of fracture was so slight that a hand lens had to be used to see the broken surface.

From inquiries it appears that the Rivière type of needle cannot be obtained without this form of graduation, which is regrettable because of its otherwise general usefulness. Each groove appears to be a situation of weakness. It is, however, possible that the main contribution toward the accident may have been my own in that by keeping too close to the upper border of the rib below the intercostal space concerned I imposed an unnecessary strain upon the cannula by causing an upward bending deflection at the point where it rode over the edge of the rib.

Reviews.

BIOLOGICAL CHEMISTRY FOR THE CLINICIAN.

The second edition of Dr. Ivan Maxwell's "Clinical Biochemistry"[1] brings within a handy compass from a wide range of sources practically all the biochemical investigations likely to be of use in the diagnosis, prognosis and treatment of disease and is welcome to laboratory workers,

[1] "Clinical Bio-Chemistry," by Ivan Maxwell, M.D., B.S., M.Sc., B.Agr.Sc.; Second Edition, 1930. Melbourne: W. Ramsay. Demy 8vo., pp. 270.

since it will save them reference to a diversity of authorities. The book is soundly planned on modern orthodox lines—discussion of physiological theory, method, interpretation of findings and, at the end of each chapter, references to the literature—and is essentially practical. From this aspect the book is quite beyond reproach, since the methods included have stood the test of more or less extended trial all over the world. It is well here to emphasize, as does the author in dealing with creatinine metabolism, that some methods do not give an accurate reflex of physiological conditions, though the clinical value of the arbitrary standards they set is not invalidated thereby. As an example, Benedict's method in our hands gives consistently high readings for blood uric acid. Perhaps Dr. Maxwell's method is also responsible for his novel statement that Melbourne subjects give higher readings than those elsewhere. We are led to believe that Professor Young's school will shortly publish on this heretofore rather unsatisfactory subject. Until then and in view of the work of Zwarenstein and the Warsaw school, Dr. Maxwell's remarks on uric acid and creatinine metabolism must be taken with a little reserve.

It is a pity basal metabolism is left out, since here is a subject in which racial characters may cause us to recast our standards.

The expressed intention of the book is to provide a groundwork for students and it was recently explained in the columns of this journal by Professor W. A. Osborne and Professor W. J. Young that Dr. Maxwell's book is a companion volume to theirs on practical biochemistry. Dr. Maxwell's intention is admirably fulfilled, though it is hoped it will not make biochemistry seem too easy. The biochemist must first be a chemist and know intimately the substances he deals with and the pitfalls of faulty reagents. Advanced workers, clinical and laboratory, will find only a framework, certainly a stimulating one, for wider knowledge to be found for a start in the literature quoted in the chapter endings. The wise clinician should consult this, since much of Dr. Maxwell's discussion is necessarily condensed. For instance, he has had to ignore Jackson's attractive theory of the histogenesis of urinary casts; his statement of the truism that insulin reduces blood sugar evidently just preceded the publication of some good work on the mechanism of this reaction; the theory of duodenal reflux neutralization of gastric juice is not given the subordinate place it deserves in view of the work of Hansman and MacLean.

This is a good little book which the publisher has turned out very agreeably, though the careless proof revision gives the work a certain lack of finish. The index is excellent.

CHRISTIAN SCIENCE.

The Right Honourable H. A. L. Fisher has written a most attractive book entitled "Our New Religion."[1] He has dedicated it "without permission to those very able publicists who, by making the *Christian Science Monitor* one of the best-informed journals of our time, have prompted at least one of their readers to ask himself the question, 'What, then, is Christian Science?'"

We would state at once that this is a book that medical practitioners will enjoy. It is extremely well written. The author has a keen sense of humour, his methods are direct and his conclusions are unassailable. Christian Science, so-called, is a force to be reckoned with and many medical practitioners must have asked themselves, as Fisher did, "What, then, is Christian Science?" This book supplies the answer. Medical practitioners have something to learn from this esoteric cult.

The author divides the book into three parts: "The Propheteſs," "The Creed," and "The Church." He describes Mary Baker in her childhood as suffering from "hysteria mingled with bad temper." He traces her development. He tells of the association with Quimby. He describes "the miracle of the fall at Lynn," when "the propheteſs," at that time Mrs. Mary Patterson, fell on the ice. He contrasts the clinical picture of the patient at the time of her fall with the impression of the fall communicated twenty years later to the world by the patient in her capacity as founder of Christian Science. Mary Baker who became successively Mrs. Glover, Mrs. Patterson and Mrs. Eddy, was a remarkable woman. The author pays a tribute to her genius for finance and shows how she built up a great financial institution with herself as the sole controlling authority, how she knew when to emphasize one aspect of her work and when to discard another that had served its purpose. But it is unnecessary to go further. The following extract is worth quoting:

> Why, then, it may be asked, does any sensible or intelligent being profess Christian Science? What is there in this extravagant form of idealism which attracts persons who are not, ordinarily speaking, regarded as cranks, and leads them to conclusions so repugnant to the general experience and wisdom of mankind? The answer is that amid much which is fantastic and false there is a central core which is true. While it is false that illness is unreal, it is true that some illnesses are caused, and other aggravated, by mental conditions which may be removed by spiritual cures. While it is false that no mental cure can be effective save when it is administered by a Christian Scientist, it is true that the serene confidence and composure of the Christian mystic may be a valuable element in the equipment of a spiritual healer. That courage and faith are powerful auxiliaries in the battle for health, and that these qualities may be communicated to the patient by a wise friend in whose nature they are already implanted, are propositions which we are all prepared to accept.

We are grateful to the author; he has given us a literary treat, amusement and instruction.

[1] "Our New Religion: An Examination of Christian Science," by the Rt. Hon. H. A. L. Fisher, F.R.S.; 1929. London: Ernest Benn Limited; Sydney: Angus and Robertson. Demy 8vo., pp. 191. Price: 7s. 6d.

Notes on Books, Current Journals and New Appliances.

RAMBLING TALES.

Sir John Bland-Sutton has written a book called "The Story of a Surgeon."[1] From the title the reader expects something in the way of a continuous narrative, if not an autobiography. Disappointment awaits him. The book is a series of pen pictures of parts of the author's life. Those who like this sort of thing will doubtless be interested, but we feel that the distinguished author has merely jotted down memories of his early days, often with little idea of making a "story." He tells of his literary education being begun by a study of the Old Testament. He states that it bred in him the critical interest in religion that stimulated him to travel in certain countries; he then adds that his love for birds and animals led him to visit other parts of the world. In the next sentence he tells of his being on deck with the captain and of being fortunate enough to witness an extraordinary shower of meteors in August, 1922, at 2 a.m., whilst entering the mouth of the Amazon. A rambling tale of no consequence! No doubt the captain's presence enhanced the wonders depicted in the heavens. From this he jumps back to his childhood's days and states: "Occasionally I stayed with my mother's relations in London." We presume he means "relatives." And so on! In his description of animals and zoological phenomena the author is interesting, but he rambles on and on in a disjointed way. Had he called his book "Reminiscences and Ramblings of a Surgeon," it would have been different. As it stands, the book is indifferent.

[1] "The Story of a Surgeon," by Sir John Bland-Sutton, F.R.C.S., with a preamble by Rudyard Kipling; 1930. London: Methuen and Company Limited. Demy 8vo., pp. 216, with twenty-eight illustrations. Price: 12s. 6d. net.

The Medical Journal of Australia

SATURDAY, OCTOBER 18, 1930.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

A MATTER OF HONOUR.

THE responsibilities of a psychiatrist are not limited to the certification of a person as insane nor to his treatment after admission to a mental hospital. He may be called upon to express an opinion as to sanity and may be asked to state his opinion in writing. He may be called upon to determine the ability of an accused person not only to appreciate the significance of a criminal act—to discriminate between right and wrong—but also to refrain from the commission of an antisocial act. He may also find himself faced with the problem of determining the capacity of a person to make a will, to transact ordinary business, to enter into ordinary contracts or to contract marriage. To have a first class knowledge of mental disease, to understand the vagaries of the human mind and to recognize the springs of peculiarities in human behaviour call for much study and many years of experience. The members of the medical profession recognize the highly specialized nature of the psychiatrist's calling and give him due honour. The non-medical sections of the community look on the psychiatrist as dealing with intangible matters—he is different from an ordinary doctor—he is to some of them a person to be feared in that he has, so it appears to them, an insight, perhaps an uncanny insight, into the hidden recesses of the mind and that his decisions may in certain circumstances lead to detention in a mental hospital. The ethical standards of the psychiatrist are, of course, the same as those of any other member of the medical profession. Since psychiatrists must always appear as beings *sans peur et sans reproche*, all sections of the community, medical and non-medical alike, cannot do other than regard seriously statements recently reported to have been made by a judge of the Supreme Court of New South Wales.

The statements referred to are reported in *The Sydney Morning Herald* of October 4, 1930, as having been made by Mr. Justice Davidson in the Probate Court on the previous day when he delivered judgement in what is known as the "Massy will case." According to *The Sydney Morning Herald* a caveat against probate had been lodged by John Eyre Massy, testator's brother, who opposed on the ground of undue influence by the widow. John Llewellyn Sands and Jessie Hilda Massy (widow), executor and executrix of the will, had asked that the caveat should be set aside and probate granted.

With the outcome of this suit we are not in the least concerned. What does concern us is the comment reported to have been made by the Judge as to one of the witnesses, namely, Dr. Chisholm Ross. He dissected the latter's evidence and is reported as having concluded by saying: "I can only say that when a person sets himself up as a mental specialist and acts as Dr. Chisholm Ross has done in this suit, he is, to my mind, nothing less than a menace to the public and a disgrace to his profession." This is, of course, a very strong and a very definite statement for a judge to make. Dr. Chisholm Ross has for many years been held in high regard by medical practitioners throughout Australia; many hundreds of medical graduates of the University of Sydney received instruction in psychiatry at his hands when he occupied the lectureship in that subject, learned to appreciate his erudition and profited by the high ethical standard that he set. Dr. Ross is also official visitor at several of the mental hospitals in the State. We realize that it is unwise to make general

deductions from excerpts from a judgement as published in a newspaper, but the Judge's conclusion, as reported above, calls for further action. The occasion, of course, is privileged and Dr. Chisholm Ross has no opportunity to reply. His honour has been impugned and he must be given an opportunity of clearing his reputation. The *Medical Practitioners Act* provides the machinery for an appropriate inquiry into the statements made concerning Dr. Ross upon which he would be afforded the opportunity of defending himself, and in the circumstances the Judge, or failing him, the Minister for Health, should direct the institution of such an inquiry.

Current Comment.

SIMPLE ACHLORHYDRIC ANÆMIA.

In regard to the nomenclature, ætiology and significance of the anæmias there exists a sad state of confusion. Any discoveries of hitherto unrecognized variations in structure, frequency or appearance of the corpuscles lead but to differentiation of anæmias into further types, and confusion becomes worse confounded. The whole subject cries out for revision, but this cannot be worthily and usefully done without some conception of ætiology and the pathological processes involved. The smug complacency of the individual who is content with his knowledge of the anæmias and who seeks no further than a tooth apex or an infected tonsil in his ætiological investigations cannot be too strongly condemned.

The very existence of disease of the blood may be questioned; anæmia should be looked upon rather as a manifestation of bodily disease or as a reflex of a disordered metabolism. After all, the blood is but a vehicle and not a living tissue in the usual meaning of the term, Professor A. E. Boycott notwithstanding. How, then, may it be the seat of disease? It may be contended that the blood is not and cannot be the seat of disease, but obviously the structure of its cells, the chemical constitution of its plasma and the efficiency of its function may be gravely affected by errors of metabolism, however produced. Carcinoma, bothriocephalus infestation, sprue and pregnancy may be associated with an anæmia cytologically indistinguishable from pernicious anæmia, yet, because of the obscurity of its ætiology, pernicious anæmia is termed a "primary" anæmia.

The association of achlorhydria with pernicious anæmia has long been recognized, but only within recent years has special attention been drawn to the frequent occurrence of a simple anæmia without obvious cause in individuals affected with achlorhydria. The condition which he terms "simple achlorhydric anæmia," has recently been carefully described by L. J. Witts.[1] This anæmia is most frequent in middle and later life and is far more common in women than in men. The anæmia is of the chlorotic type, there is no evidence of increased blood destruction, and the Van den Bergh reaction is absent. There is evidence in the bone marrow of increased erythroblastic activity, and normoblasts are more numerous here than in health. The disease is rarely fatal, but has little tendency to spontaneous recovery. It is apparently unaffected by the administration of liver. The coexistence of glossitis is common. Splenomegaly is occasionally observed; it is more apt to occur when the anæmia is severe. Chlorosis is readily distinguishable, as it occurs most frequently during adolescence, is not associated with achlorhydria, tends to spontaneous recovery and is not likely to recrudesce in middle life. Witts believes that simple achlorhydric anæmia is more common than Addison's anæmia and is a fruitful source of chronic invalidism in middle-aged women. A notable feature is the effect of pregnancy, which aggravates an existing anæmia and sometimes precedes the onset. In some instances Witts has observed that there has been a history of *post partum* hæmorrhage. Functional disturbances of the reproductive organs are common. Achlorhydria persists despite treatment and apparent cure of the anæmia. He points to the existence of transitional forms linking simple achlorhydric anæmia and Addison's anæmia, to the occurrence of secondary anæmia sometimes in subacute combined degeneration of the spinal cord, to the occasional low colour index of Addison's anæmia *et cetera*, and ventures the opinion that the two conditions are in some way related and due to the same cause, namely, achlorhydria. The grounds for his belief are not apparent. He goes on to say that achlorhydria in males is apt to lead to a macrocytic anæmia, in females to anæmia of a microcytic or secondary type. He states that on rare occasions simple achlorhydric anæmia, with the passage of time, may actually change into anæmia of the Addisonian type. He draws attention to the well known tendency to the familial occurrence of Addison's anæmia, but states also that simple anæmia is apt to occur in the same families. He quotes authorities to assist his argument. He discusses the very interesting Plummer-Vinson syndrome. Briefly, this consists of secondary anæmia and dysphagia and, usually, glossitis and achlorhydria. According to Hurst, the dysphagia is due to achalasia of the pharyngo-œsophageal sphincter caused by degeneration of the vagus nerve endings in Auerbach's plexus. Witts believes that simple achlorhydric anæmia and the Plummer-Vinson syndrome are closely akin and bear much the same relationship to each other as do Addison's anæmia and subacute combined degeneration of the cord.

Witts has thrown light from a new angle on the condition which he terms "simple achlorhydric

[1] *Guy's Hospital Reports*, July, 1930.

anæmia," and his contribution might prove of considerable value in the study of the anæmias generally. The relationship of achlorhydria to pernicious anæmia has been discussed in these pages on many occasions and was last discussed in the issue of April 26, 1930, in connexion with a communication by J. M. Vaughan. The bacterial origin of pernicious anæmia is the hypothesis that most easily fits the known facts. At the same time it might be argued that both pernicious anæmia and achlorhydria are due primarily to the same metabolic disturbance. The coexistence of achlorhydria with secondary anæmia of middle-aged women may be merely fortuitous, at any rate in many instances. Of course, the occurrence of secondary anæmia in women affected with decrease or absence of gastric hydrochloric acid is quite common, but such a condition scarcely appears to be worthy of an individual title. The woman affected with achlorhydria often is of the thin visceroptotic type, and suffers from chronic intestinal stasis. By the time she has reached middle age she has probably had several pregnancies with consequent weakening of already hypotonic abdominal musculature and aggravation of visceroptosis and constipation. Who, then, is more likely to become affected with secondary anæmia? Achlorhydria, when not due to purely local causes, appears to be only one feature of a disturbance of metabolism or a characteristic of a physiological type. As such its importance cannot be overlooked and it must be remembered that its ætiological relationship with anæmia has not been definitely proved.

DIETARY CORRECTION OF OBESITY.

At the present time, when there is a vogue for slimness, the subject of obesity and diet is most popular among the laity. This popularity is the occasion of much thought and sometimes of much worry to physicians. The subject was discussed in this journal in September, 1927, in a series of articles by Blackburn, Bullmore, Holmes à Court and Hansman. Readers will find the articles by these four writers worthy of reference and of careful study. It is not intended to reopen the whole question in the present instance, but to call attention to a communication by J. M. Strang, H. B. McClugage and F. A. Evans.[1] These authors begin with the statement that in the treatment of obesity it is necessary to appreciate the fundamental facts of the metabolism of the obese and to adapt the generally accepted principles of dietetics to meet the needs of the physiological anomaly. This, of course, is of basal importance and an endeavour should always be made to discover some endocrine cause for the condition. The authors state that in the absence of convincing evidence pointing to a metabolic economy in the obese, they are forced to the opinion that fat deposits result solely from an excess of energy intake over energy output. The only method for the dissipation of this energy reserve is its combustion in the usual manner. The diet designed by them is claimed to supply the smallest possible intake and to preserve the maximum output. It consists of sufficient protein to maintain the body requirements together with enough vitamins and salts to prevent the occurrence of complications resulting from deficiencies. The only carbohydrate and fat given are those required by the protein and inseparable from accessory food substances. One hundred grammes of lean steak were the backbone of each of the two largest meals. Egg white was used to balance the daily protein requirement. On alternate days the patient received either one hundred cubic centimetres of whole milk or of orange juice and fifty cubic centimetres of actively growing yeast. One glass of Kolok water was given every day. Later 5% vegetables were given. The patient drank 2,500 cubic centimetres of water every day. The diet averaged only 360 calories a day and these were derived from fifty-eight grammes of protein, fourteen of carbohydrate and eight of fat. Thirteen patients lost on an average 27·2 grammes or 0·6 pound per day for fifty-nine days. No untoward reactions were noted in spite of an early nitrogen loss and of a theoretical ketogenic-antiketogenic ratio of 3:4. Patients were maintained on this diet without complications for six months. In treatment of obesity sudden reduction in weight should be avoided and, if treatment of so drastic a nature as that described by the authors is attempted, the patient must be carefully watched. There is always a danger of acidosis. Strang and his collaborators state that when their diet is used, fat is burned up in the usual way. If fat is to be burned up in the usual way, a sufficient amount of carbohydrate must be available. For this purpose some investigators have initiated their treatment with the administration of glucose and alkalis.

[1] *The American Journal of the Medical Sciences*, May, 1930.

AUSTRALIAN MEDICINE AND THE GREAT WAR.

The first volume of the "Official History of the Australian Army Medical Services, 1914-18" is shortly to be published. The compilation of this work has been arranged by the Commonwealth Government and it has been edited by Colonel A. Graham Butler, D.S.O., V.D. The publication of this book is an event of the first importance, not only to the medical profession of Australia, but to the whole community. It is hoped that it will be possible to publish an adequate notice of the book soon after its appearance. In the meantime it should be noted that a prepublication offer has been made to the members of the medical profession. Those who order the book from the Director of the Australian War Memorial, Box 214 D, G.P.O., Melbourne, will obtain it at nineteen shillings per volume instead of twenty-one shillings and sixpence. Volume I should be ordered before November 15, 1930.

Abstracts from Current Medical Literature.

DERMATOLOGY.

Testicular Extracts in the Treatment of Ulcers.

FILIPPO RHO (*Journal of Tropical Medicine and Hygiene*, April 15, 1930) discusses the treatment of ulcers by the local application of testicular extracts and quotes a personal experience. In the year 1890 Aievoli published a paper in which he pointed out the value of the local application of thin slices of fresh testicles of rabbits and guinea-pigs in the treatment of chronic ulcers, especially varicose ulcers. Various observers in Italy, France, Germany, England and America have since testified to the value of testicular extracts in the treatment of these conditions. The author suffered from an ulcer on the right leg, the result of an injury. At the end of six weeks' treatment no improvement was evident. He then had recourse to a glycerinated extract of testicle, called "Architasi," which is prepared by Serono. He applied a few drops to the sore on every alternate day, covering the bare area with gauze on which some ointment of zinc oxide and ichthyol had been spread. The ulcer healed in less than a fortnight, leaving a smooth whitish scar with pigmented margins. The author suggests that this method might prove of value in the treatment of some of the ulcers which are encountered so frequently in tropical countries.

Epidermomycosis of the Feet.

K. P. A. TAYLOR (*United Fruit Company Medical Department, Eighteenth Annual Report*, 1929) remarks on the unsatisfactory results of treatment by the usual methods of ringworm of the feet, and suggests a line of treatment which proved successful in a personal experience of epidermomycosis of the feet and which met with equally good results in six other instances. The treatment consists in spraying the parts affected with ethyl chloride until blanching of the skin is produced over an area extending beyond the periphery of the lesion for a distance of 0·5 centimetre. Blanching should be maintained for a period of one-half to one minute. The application should be repeated every day or, on the plantar surface, twice a day. Two to six or more applications may be required. Loose skin and overhanging margins of epidermis are trimmed away in order to render the fungus more vulnerable to the attack of the ethyl chloride. The treatment is contraindicated if an eczematous dermatitis or acute inflammation is present.

Arsenical Dermatitis.

JOSE ALBERTO LOPEZ (*United Fruit Company Medical Department, Eighteenth Annual Report*, 1929) reports a case of arsenical dermatitis occurring in a labourer employed in antimalarial work. Paris green mixed with road dust or saw dust has been used as a larvicide for several years on the Truxillo Railroad Company's plantations to prevent the breeding of anopheline mosquitoes. The mixing is usually done in a house or shed where there is protection from the wind, and it is impossible for the labourer to avoid inhaling a quantity of dust and becoming covered on the extremities and exposed parts of the body with a thin layer of dust. The mixture is broadcast over water by hand. The proportions employed are usually one part of Paris green to one hundred parts of excipient. The occurrence of arsenical dermatitis among labourers using the powder is a rarity. A labourer, seen by the author, suffered from a severe dermatitis of the exposed parts. The face and neck were swollen, red and indurated, and presented small papules and a few pustules and a slight degree of fine desquamation. A similar condition was observed on hands, forearms and feet, and on a narrow strip across the lower part of the abdomen where the shirt was usually open and loosely adjusted. Three days' absence from duty resulted in a remarkable improvement. Four days after the patient's return to duty, however, the dermatitis recurred with all its former severity. A transfer to other duties led to apparent permanent cure. The author suggests the possibility of sensitization to arsenic in these cases.

Subcutaneous Cysticercosis Cellulosæ in Man.

CHUAN-KUEI HU, OO-KEK KHAW AND C. N. FRAZIER (*Archives of Dermatology and Syphilology*, May, 1930) report five cases of *cysticercosis cellulosæ* occurring in the subcutaneous and muscular tissues. The characteristics of the cysts, when they are located in subcutaneous tissue, are such as to make their identification possible by physical examination alone. They are as freely movable as a foreign body, of elastic hardness, not tender and generally multiple. The average size was one centimetre in diameter. Cutaneous sensitization tests performed on two of the patients with the fresh fluid from a cyst gave no reaction.

Cutaneous Manifestations of Heredity.

C. M. WILLIAMS (*Archives of Dermatology and Syphilology*, May, 1930) in a paper dealing with the influence of heredity on disease discusses a number of cutaneous peculiarities in the light of various Mendelian factors. Albinism and the mechanism of transmission of eye colour are dealt with. It is explained that a character that has not been observed in the ancestry for generations, is definitely inherited according to known laws. Certain conditions, such as skin colour, hair and eye colour, various deformities of the nails, *epidermolysis bullosa* and hereditary hæmorrhagic telangiectasia, all of which are hereditary and apparently independent of disease of other organs, are probably due to direct inheritance based on a few simple factors. In the second group of manifestations it is difficult to determine whether the visible inherited peculiarity, such as excessive or deficient growth of hair, is due to a peculiarity in the skin itself or to some alteration of function of the endocrine or of some other system. Chief in this group are xeroderma and other allied conditions. Ichthyosis of various grades, as *keratosis pilaris*, oxychogryphosis, *keratosis palmaris et plantaris*, are all forms of an abnormal keratinization, and the character the deformity takes depends on another inherited factor. The factor which is inherited may not concern the skin *per se*. It is probable that in many families there is an inherited tendency to endocrine dysfunction and that the cutaneous manifestation will vary with the gland involved and with its excessive, deficient or perverted action. In the author's opinion psoriasis is probably an inherited disease and there is a congenital mode of reaction of the skin which may be made apparent by different endogenous and exogenous agents after the skin has been sensitized by endocrine dysfunction. In the cases cited by Bernhardt, in two families the disease appeared in both parents and all children; it may be assumed that the factor was dominant and that at least one of the parents was homozygous or that the factor is recessive; in these circumstances the parents and all the children must be homozygous. There would appear to be in all hereditary cutaneous peculiarities one factor for the production of the condition and one for its distribution. On the question of cancer the author holds that there is sufficient evidence of an inherited susceptibility and that clinical and pathological evidence point to a dominance rather than to recessiveness in the inheritance of this susceptibility. The importance of heredity in the allergic group of diseases is well recognized and in these the tendency inherited is probably a simple dominant. The mode of inheritance is not known, but what is inherited is a capacity to become sensitive to certain foreign substances, not necessarily protein, and the transmitted capacity is not specific. With regard to the resistance to infection, both botanists and zoologists have noted the inheritance of variations in susceptibility. Resistance is not entirely a specific quality. In regard to syphilis, if the course and manifestations of the disease depend, partly at least, on the allergic or non-allergic character of the individual and if allergic irritability is an inherited quality, it follows that in syphilis the mode of reaction depends on the hereditary constitution of the individual and that the tabetic patient belongs to the anallergic group.

Hepato-Recurrent Syphilis.

S. S. GREENBAUM (*Archives of Dermatology and Syphilology*, May, 1930) reports a case of jaundice occurring in a syphilitic patient three and a half months after four injections of "Neoarsphenamine." The infection proved fatal and the *post mortem*

findings were those of acute yellow atrophy of the liver. Intravenous injections of sodium thiosulphate, dextrose and sodium iodide and a single intramuscular injection of bismuth did not stay the progress of the disease. The condition was probably recurrent syphilis of the liver and the author is inclined to agree with Milian who insists on the continued use of the arsenical compounds in post-therapeutic types of icterus or in those developing from two weeks or more after the last injection.

UROLOGY.

X Ray Therapy in Prostatic Cancer.

G. G. SMITH AND E. L. PIERSON (*Journal of Urology*, March, 1930) publish their results in twenty-five cases of prostatic carcinoma. High voltage X ray treatment was used and the authors conclude that: (i) Urinary symptoms must be treated by suitable local measures or by surgical operation, as they are not improved by X rays. Likewise, the pain due to obstruction or cystitis is not affected by radiation. (ii) X rays, however, will in most instances relieve the other types of pain due to the pressure exerted by the original growth or by its metastases. (iii) The duration of this relief is variable, but lasts from one to six months after such treatment. (iv) In some instances the malignancy of the growth appears to be reduced. (v) Three or four series of treatments, given at intervals of two to three months, should be given, but no more.

Treatment of Various Types of Hydronephrosis.

M. MARION (*Journal d'Urologie Médicale et Chirurgicale*, June, 1930) considers that when an abnormal vessel is found to be the cause of a hydronephrosis, the vessel should be divided. Division and reimplantation of the ureter are now known to give bad functional results in the long run, while division of the vessel does not cause any real damage in the area of kidney supplied or drained by it. Secondly, those hydronephroses caused by definite renal ptosis are to be treated by nephropexy. In order to avoid the bad results so often complained of, the technique must be of such a kind as to effect an actual replacement of the organ in its natural high position, well up under the shelter of the twelfth rib. The well known capsule flap operation of Marion achieves this result, but the sutures employed should be non-absorbable and should be inserted above the tenth, eleventh and twelfth ribs respectively, and should pass horizontally through these intercostal spaces, pleural cavity and diaphragm, in order that the kidney may be elevated to the proper height. The third group of cases are the most difficult; here a definite hydronephrosis, even though sometimes it is small, exists, but there is no apparent cause discoverable, either by pyelography or at operation. Denervation of the renal pedicle has been proposed for this condition, but while this operation relieves the pain, it fails to arrest the progress of the hydronephrosis. Moreover, it is not yet certainly known whether damage results from cutting off the trophic nerve supply. Marion thinks that the actual cause of pain in these circumstances is a slight ptosis of the kidney, not discernible by palpation or by pyelography, but producing its evil effect because of the fact that it coincides with a relative fixation of the upper part of the ureter. In addition to the performance of nephropexy, it is well to free the upper portion of the ureter from all adhesions. Since 1912 the author has operated on twenty-three patients suffering from this type of pain and by the above means has secured uniform and perfect success.

Changes in the Urinary Tract in Pyelitis of Pregnancy.

O. BRAKEMANN (*Verhandlungen der Deutschen Gesellschaft für Urologie*, 1929) has made urographic and anatomical researches in the changes, particularly in the course of the ureter, that take place when the ureter is obstructed somewhere about the brim of the pelvis during the pyelitis of pregnancy. The chief point apart from the dilatation of the calyces, pelvis and ureter, is that the latter canal when obstructed tends to elongate. In order to accommodate itself within the anatomical space at its disposal, the elongating ureter becomes bent and even twisted. The only part at which it has complete freedom is in its upper third where it is running forward through the loose fatty tissue of the renal fossa to reach the back of the peritoneum, and it is definitely to be noted that it is only in this upper third that kinks, twists or loops are formed. In the lower part of the abdominal course of the canal a simple lateral curve generally forms. In this part the ureter lies on the flat anterior surface of the psoas muscle and is bound down here by the parietal peritoneum and so is prevented from bending forwards. It is prevented from bending medially by the strong tubes of the *vena cava* or aorta, as the case may be, and so is compelled to fall laterally and then posteriorly over the steep lateral surface of the psoas muscle until it reaches the iliac fossa. From here, as it goes downwards, it climbs gradually up again on to the anterior surface of the psoas muscle, bending almost to a right angle as it does so, and then travels medially towards a fixed point on the pelvic brim, whence it falls into the *pelvis minor*. In ureterograms the posterior curvings of the canal cannot be appreciated, but the lateral and medial curves are well seen. The more detailed work has been done on the cadaver.

Bilateral Renal Calculi.

N. F. LESCHNEW AND D. E. LEVANT (*Zeitschrift für Urologie*, January, 1930) plead for more conservatism in the problem of the surgical treatment for the relief of bilateral renal calculi. It is very often better to withhold operative intervention. Renal calculi are present on both sides in at least 25% of all instances. In young people with aseptic or only slightly infective calculi some authors advise operating on both kidneys at the one sitting, but the writers think the unilateral operation is safer. Where urea-splitting organisms (staphylococci, *Bacillus proteus*) are present, it is better not to operate unless the indications are vital and urgent, as under such conditions early relapse is likely to occur. The same remark applies to cases of infected phosphatic stones. Bilateral nephrolithiasis must be regarded as a serious systemic disease and operation must not be considered when the general condition is below normal or when examination of the better kidney does not reveal a perfectly normal efficiency. When the disease is of long standing operation may only be expected to shorten the patient's life. Very large coral-shaped calculi and multiple calculi in the renal parenchyma constitute a contraindication for operation, even when the general condition is good and the renal efficiency satisfactory.

Renal and Ureteric Lesions Secondary to Male Adnexal Disease.

A. VON LICHTENBERG (*Journal of Urology*, July, 1930) recalls that inflammatory and obstructive disease in the upper urinary tract in males in many instances is really secondary to inflammation occurring primarily in the adnexa (prostate gland and seminal vesicles). The adoption of this conception of "system pathology" will not result in the divorce of the various parts of the genito-urinary system in the male from one another. In such disease this point of view demands that therapy should begin in the primarily diseased adnexa, even if the secondary changes higher up have already achieved pathological importance and independence. The usual sequence is that prostatitis and seminal vesiculitis lead to periprostatitis and perivesiculitis; these changes lead to a plastic inflammation of this part of the pelvis which then induces ureteritis and occasionally stenosis of the lowest part of the ureter. From this follow obstruction and dilatation of the renal pelvis and ureter, often with tortuosity or kinking of the latter tube and sometimes chronic interstitial nephritis or even suppuration of the kidney. A ureteric stenosis must be relieved, but the diseased adnexa must also be treated or else the therapeutic success will be only short-lived. Conservative treatment of the adnexa consists of diathermy of the adnexa and intravenous injections of mercurochrome, "Acriflavine," "Salvarsan" or urotropine. When surgical treatment is necessary, it consists of cystoscopy, incision of the ureteric orifice by diathermy, Belfield's operation of vasotomy, perineal prostatectomy, sacral vesiculectomy and various appropriate operations directed towards gross lesions of the upper urinary tract.

Special Articles on Diagnosis.

(Contributed by Request.)

XVI.

CŒLIAC DISEASE.

ANY definition of cœliac disease must be unsatisfactory until we know the actual cause of the essential feature, namely, the failure of the body to utilize fat. The cardinal features of this disorder are enlargement of the abdomen without signs of organic disease, persisting or relapsing diarrhœa with pale, fatty stools and retardation of physical growth and development.

The age at onset is in the majority of cases the later part of infancy, from the age of nine months to three or four years. The disease seems to occur more frequently in girls than in boys. The onset is usually insidious, frequently with a history of preceding gastro-intestinal disturbance, unsuitable feeding, or an attack of dysentery. The child becomes irritable and fretful, begins to waste and the stools assume the characteristic appearance. The illness may last for two or three years, but when recovery takes place it seems to be complete.

The wasting is most pronounced in the limbs, the face often appearing plump and rounded. The weight for age is much below normal, but the weight for height is more nearly normal, showing the failure of the child to grow. The abdomen is in marked contrast to the wasted limbs. The abdominal wall, except in the most severe cases, is not particularly wasted. The enlargement is due partly to distension and partly to the weakness of the muscles. Neither liver nor spleen is enlarged.

The stools are large, pale, offensive and unformed. They may have a lustrous appearance due to the excess of fat. Usually three or four stools are passed in the twenty-four hours and these may occur mostly at night. The excess of fat in the stools shows that fat wastage is out of all proportion to that of other foods and it persists when the rest of digestion is normal. Even on a strict low fat diet the fat in the stools remains abnormally high. The fat is invariably well split, the neutral fat being well within normal limits. During the course of the disease the stools may temporarily change their character and become brownish or green with mucus. This is evidence of intestinal irritation set up probably by the excess of fat in the stools. No evidence of pancreatic disease has been found *post mortem* nor by examination of the stools. That calcium metabolism is interfered with is shown by the fact that the bones show osteoporosis and if the disease persists to the age of seven years or thereabouts, rickets of the low calcium type may develop. Also tetany is not an uncommon occurrence during the illness. There is marked muscular hypotonia which interferes with the child's ability to walk.

Laboratory Investigation.

The generalization may be made that normally the amount of fæcal fat should not exceed one-third of the dried fæces and that of this fat not more than one-third should be present as neutral fat. In cœliac disease, if the stools are examined for fat, the amounts found are from 40% to 80% of the dried fæces. These fats are well split, neutral fats being as a rule well under 25% of the total fat. In the severe cases the split fats are largely in the form of fatty acids. As improvement occurs, the soaped fats increase and the fatty acids diminish.

The blood shows usually a slight anæmia. The chief change is a deficiency of hæmoglobin.

Moncrieff and Payne have recently found an increase in the blood fat in a series of cases of cœliac disease. If this proves to be a usual finding, it would suggest that the disorder is not due to a malabsorption of fat, but to some other derangement of fat metabolism.

Differential Diagnosis.

Abdominal tuberculosis may produce fatty diarrhœa by inflammation and ulceration of the intestines or by lacteal obstruction due to pressure of tuberculous glands. This condition is excluded by the absence of reaction to the tuberculin test (either von Pirquet's or the intracutaneous test), the absence of enlarged glands or other masses in the abdominal cavity and of other signs of tuberculosis.

Chronic Intestinal Indigestion.

In chronic intestinal indigestion there is failure in the digestion of all kinds of foods and the hastening of the stools through the intestine and disease of the intestinal wall may be sufficiently severe to cause an excess of fat in the stools. The wasting of the patient is more severe than the lack of growth. The stools tend to show the presence of mucus and of undigested food. The digestion of all foods is impaired, that of fat with and not out of proportion to the rest. On a low fat diet the percentage of fat in the stools drops to a lower figure than in true cœliac disease. In some of these cases, if persistent, a secondary pancreatitis may develop and presumably a pancreatic steatorrhœa might arise from this cause. Still and others have reported such cases.

Pancreatic Steatorrhœa.

Pancreatic steatorrhœa is very rare in infants and young children and when it does occur, is usually congenital. The stool of pancreatic disease is large, pale, offensive and oily. It contains a great excess of fat and its diagnostic feature is in the distribution of the fæcal fat. The amount of neutral fat is usually much above one-third of the total fæcal fat, that is, there is a failure in the fat-splitting.

The examination of the fat in the stools of a child with cœliac disease made by Harrison and Sheldon gave the following results: Total fat formed 58·95% of dried fæces. Of this fat 88·3% was split fat and 11·7% was unsplit. In a child under my care suffering from congenital pancreatic steatorrhœa with infantilism, the result of the examination of the stools made by Dr. Eva Shipton was as follows: Total fat formed 60% of dried fæces. Of this fat 53·8% was split fat and 46·3% was unsplit.

In this condition the neutral unchanged fat shows itself in the stool as separated oil or grease which congeals on cooling. This type of stool does not occur in cœliac disease.

Summer Diarrhœa.

Under the heading of summer diarrhœa are included the two main types of infectious diarrhœa which occur during the summer months, namely, gastro-enteritis and bacillary dysentery.

Gastro-Enteritis. Gastro-enteritis, called sometimes in severe cases *cholera infantum*, is an extremely fatal disease, especially in infants under one year of age. The onset is usually sudden, with vomiting and diarrhœa accompanied by fever. The stools are frequent and green, containing much mucus, and in severe cases may be watery and almost colourless. Often towards the end, if the disease is running a fatal course, the stools become a bright orange-yellow colour. The vomiting may cease soon after the onset of the diarrhœa or may continue in spite of all treatment until death occurs. Frequently, if vomiting persists, the vomitus has a coffee ground appearance. This is an ominous, but not necessarily a fatal sign. The blood comes from oozing of the gastric mucous membrane which on *post mortem* examination appears congested and reddened without ulceration.

The disease may be divided into three types, the mild, the severe and the fulminating.

In mild cases, if proper treatment is adopted, the patients recover in a few days or a week, but if they are untreated or improperly treated they may pass into the severe type. The patients suffering from severe gastro-enteritis become dehydrated and at any period of the disease severe toxæmia may occur. Petechial hæmorrhages and œdema of the limbs are not uncommon in the prolonged cases.

In the fulminating type of gastro-enteritis the child suddenly becomes acutely ill, with high temperature and an extremely toxic appearance. There may be little or no dehydration. The child is comatose, the face grey, with sunken eyes. The mucous membranes of the mouth

and the lips are bright red, in marked contrast to the greyish pallor of the face. The breathing is rapid, vomiting occurs and diarrhœa may be present, although death may occur without the appearance of abnormal stools in twenty-four or thirty-six hours from the onset of the disease.

In regard to the differential diagnosis of gastro-enteritis, dyspepsia due to unsuitable feeding may be accompanied by diarrhœal stools and vomiting. The careful consideration of the general condition of the child and the history of the feeding should disclose the cause of the digestive upset.

Parenteral infections, especially in infancy, are accompanied by diarrhœal stools, vomiting and general toxæmia and without a thorough examination of the child mistakes in diagnosis may be made.

Otitis media is a common cause of such symptoms and may be accompanied by severe intoxication. The thorough examination of the naso-pharynx and the ears should enable one to make a correct diagnosis.

Pyelitis is often accompanied by marked toxæmia resembling that of gastro-enteritis. There may be no signs of urinary disease noticed by the mother and the condition may be easily overlooked unless the urine is examined.

Pneumonia, bronchitis and other parenteral infections may be excluded by a complete examination of the child.

Bacillary Dysentery. Bacillary dysentery is always prevalent in the summer months, although small epidemics may occur at other periods of the year. The onset is sudden, with fever and malaise, followed by vomiting and diarrhœa. The stools are at first relaxed, greenish and mucous, followed at longer or shorter interval by evacuations composed mainly of blood and mucus. Small quantities of blood and mucus may be passed very frequently with much pain and tenesmus.

In mild cases the fever disappears, the motions become less frequent and contain more fæcal matter and in a few days recovery may be complete.

In other cases the fever and dysenteric stools may continue for some weeks and at any period of the illness dehydration and intoxication may appear.

The specific organism may frequently be recovered from the stools, but the fact that no pathogenic organism is found does not exclude the diagnosis of dysentery. The longer the disease has lasted, the less likely is the organism to be recovered.

The diagnosis is made from the clinical signs and symptoms and when possible by bacteriological examination. Any condition with fever, blood and mucus in the stools accompanied by tenesmus may be diagnosed as dysentery. There are, however, cases in which no blood appears in the stools and in which the dysentery bacillus is found on culture. Indeed, death may occur from dysentery before blood appears in the evacuations.

In order that the bacteriological examination may be successful the stool must be carefully collected and delivered for examination without delay. In this connexion I cannot do better than quote from Dr. Marjory Little, who says:

> In dealing with cases of bacillary dysentery, positive findings are most likely to be achieved if the stool is examined early in the disease and as soon as possible after defæcation.[1]
>
> The dysentery bacilli are peculiarly sensitive to the presence of acid. The above mentioned workers (Dudgeon, Urquhart and Logan) found that if early examination was not possible, the addition of an equal quantity of 3% normal sodium hydroxide to the stool greatly increased the numbers of their positive findings.

Dr. Little suggests that specimens which cannot be examined within six hours of evacuation, should be treated in this way. The following is a description of the method of collecting a specimen for examination which proved successful in the hands of Dr. Little and Dr. M. Ross.

The bowel is washed out with sterile normal sodium chloride solution. The first ten cubic centimetres returned are collected in a sterile test tube; the bulk of the fluid from the bowel is then allowed to escape and the material which is judged to be the last portion to be returned from the bowel, is also collected separately for examination. The two test tubes are despatched to the laboratory and examined immediately, if possible. The first sample is usually formed of fæcal material in saline solution and is often unsuitable for bacteriological examination. The second specimen consists as a rule of flakes of mucus floating in the saline solution and provides material favourable for bacteriological investigation. If this method of collecting a specimen is impossible, the napkin soiled by the patient should be forwarded to the bacteriologist so that he may select his own material.[2]

References.

[1] E. Marjory Little: "Dysentery: Bacillary and Amœbic," The Medical Journal of Australia, January 6, 1923, page 1.

[2] E. M. Little and M. J. Ross: "Dysenteric Conditions in Children," "Transactions of the Australasian Medical Congress (British Medical Association)," Supplement to The Medical Journal of Australia, July 5, 1924, page 472.

Margaret H. Harper, M.B., Ch.M. (Sydney),
Honorary Physician, Royal Alexandra Hospital for Children, Sydney.

British Medical Association News.

NOMINATIONS AND ELECTIONS.

The undermentioned have been nominated for election as members of the New South Wales Branch of the British Medical Association:

Heaslip, William Gordon, M.B., B.S., 1929 (Univ. Adelaide), Salamo, *viâ* Samarai, Papua.

Taylor, Clive William, M.B., B.S., 1929 (Univ. Sydney), 8, Alpha Street, Willoughby.

The undermentioned have been elected members of the Victorian Branch of the British Medical Association:

Maynard, Roy Bryant, M.B., B.S., 1930 (Univ. Melbourne), 8, Russell Street, North Williamstown, W.16.

Stonham, Franklyn Victor, B.Sc., M.B., B.S., 1929 (Univ. Melbourne), Melbourne Hospital, Melbourne, C.1.

Medical Societies.

THE CLINICAL SOCIETY OF THE MATER MISERICORDIÆ HOSPITAL.

A meeting of the Clinical Society of the Mater Misericordiæ Hospital was held at the Mater Misericordiæ Hospital, Brisbane, on July 15, 1930, Dr. E. D. Ahern in the chair.

Scirrhous Cancer of Colon.

Dr. L. M. McKillop showed a male patient, aged fifty-six years, from whom he had removed the pelvic colon and rectum by the abdomino-perineal route. The patient had been ill for some weeks with periodic attacks of cramping pain in the lower part of the abdomen, the passage of blood-stained mucus and diarrhœa alternating with constipation. Laparotomy had revealed very slight involvement of the glands in the pelvi-rectal mesocolon and an absence of secondary deposits in the liver.

The specimen removed was also shown to the meeting. It proved to be a scirrhous carcinoma of the ring type.

Carcinoma of the Rectal Ampulla.

Dr. McKillop also showed a specimen of the ulcerative type of carcinoma of the rectal ampulla, also removed by the abdomino-perineal route with excellent result.

There had been no deaths among the last six patients with rectal cancer operated on by this route by Dr. McKillop.

During the course of some remarks upon cancer of the rectum, Dr. McKillop pointed out that cancers usually arose in the alimentary canal at sites where the calibre of the alimentary canal either underwent a narrowing or a change in direction, or where one type of epithelium merged into another type. There was good reason for believing that cancer of the rectum mostly arose from the "degeneration" of preceding papillomata. Polyposis of the rectum was a definite premalignant state. Constipation in a patient with such a condition was a possible exciting factor in the change from simple polyposis to cancer.

Dermoid Cyst of the Ovary.

Dr. McKillop showed a fairly large dermoid cyst of the right ovary. This, on being opened, was found to contain a large amount of red hair, similar to the hair present on the body of the young woman from whom the specimen was removed. The cyst also contained a large amount of cholesterol.

Gastric Ulcer.

Dr. P. J. Kelly showed a male patient whose past history was that he had been operated on on April 25, 1929, for excision of gastric ulcer and gastro-enterostomy. Prior to this he had had indigestion for a number of years, with pain in the abdomen and wind about one and a half hours after meals. He had had practically no vomiting, but only a little dry retching with no relief, never self-induced. He had had hæmatemesis once, about six weeks after the operation, and no melena at any time. The pain had generally awakened him at nights and had been relieved by bicarbonate of soda or food. The pain had nearly always come on one and a half hours after food in the day or after strenuous exercise. At times the patient would have an interval of freedom from pain lasting six to eight weeks. After operation the patient had had the same symptoms, but exaggerated. He had lost 6·3 kilograms (one stone) in weight in six months and had been unable to work.

He had been admitted to hospital and put on a strict diet with immediate relief. His teeth had been removed and at present he was free from pain. He stated that he had had dietary treatment out of hospital with relief, but as soon as he began work again the pain recurred. An opaque meal had been given which showed a small six-hour residue, with apparently all the meal leaving by the stoma which was narrowed. The pylorus was narrow. Beyond the opening of the stoma the stomach did not relax at all.

Dr. A. Lee considered that a fractional test meal should be done, as the free acidity should have been reduced to practically nil.

Dr. L. McKillop thought that there was an insufficient gastro-enterostomy opening; scarring and acidity were still present.

Erythema Multiforme.

Dr. Kelly also showed a female patient suffering from *erythema multiforme*, of particular interest on account of the severe constitutional upset which it occasioned. The patient had suffered from septic tonsils with a rise in temperature. As this settled down she had broken out in an erythematous rash on both arms, both hands, shoulders and legs and on the neck. The rash had become successively papular, vesicular, pustular and had then peeled off in large crusts.

The patient had been very ill, with a temperature of 39·4° C. (103° F.) lasting over a week, then dropping to 38·3° C. (101° F.) and gradually settling down. She had had a rigor at the commencement.

The usual cause of *erythema multiforme* was generally some septic condition in the throat or body, or else it was due to some serum. In this case it was evidently due to infection in the throat. The treatment generally given was calcium lactate, sodium salicylate, adrenalin and quinine, but Dr. Kelly said he did not think drugs had any effect on the course of the disease; it ran a normal course of fourteen days. This had been a very severe infection.

Dr. Kelly also showed another patient with *erythema multiforme* in which the rash had completely gone. This case had not reached the vesicular stage. The patient, a man, had been admitted very ill with a temperature of 39·4° C. (103° F.) and had looked a typical sufferer from erythema, with the rash on both hands and feet and patches all over the chest and shoulders.

A slight tingling pain had been present and some itching in the hands and feet and the constitutional disturbance had been marked. No focus had been found in this patient and the disease had died down in fourteen days. In rare cases streptococci had been found in the blood.

Fæcal Fistula.

Dr. Milton Geaney showed a patient whom he had shown at a previous meeting in October, 1929. The patient, a male, had been admitted twelve months before with a ruptured duodenal ulcer. This had been oversewn and a tube had been put into the abdomen; a large mass had been felt in the caecum. Three months later a partial colectomy had been done and an anastomosis performed. Later a fæcal fistula had formed through the abdominal wall and the intestinal juices had started to digest the patient's own tissues, with the result that he had a hole in his abdominal wall 7·5 centimetres (three inches) in diameter. After several kinds of dressings had been used, hydrochloric acid and "Bovril" dressings had been used and the hole had begun to heal. Last time the patient was shown the hole had been 6·25 centimetres (two and a half inches) in diameter. These dressings had reduced the hole to the size of threepence or even smaller, and then Dr. Geaney had tried to dissect off the bowel and invert it with stitches, but it had broken down again. It had become closed once more to a hole admitting a knitting needle and the contents of the bowel flowed out through this and had been very distressing.

Pure carbolic had then been used to scarify the skin growing down the track and at the time of the meeting there was only a small hole that would admit the point of a needle. On some days it was quite clean, on other days there was a slight stain.

Amœbic Hepatitis.

Dr. K. C. Ross showed a patient, aged twenty-nine years, who had come from a country district two months previously with a diagnosis of pyonephrosis. For one week he had felt ill and off colour, with headaches and some pain under the right shoulder blade. On examination the patient had been deeply jaundiced and very ill, with a temperature of 38·9° C. (102° F.). Tenderness and rigidity had been present in the upper part of the abdomen.

The diagnosis had been obscure, but an immediate operation was performed, when an enormous liver had been found and a spleen which extended to the costal margin, but not below it. The blood in the wound appeared blue, but the patient was not cyanosed. Nothing else abnormal had been found. The patient had been very ill for a few days. The blood count had revealed a moderate leucocytosis. On questioning the patient further it was found that for four to five years he had suffered from severe attacks of diarrhœa with blood and mucus. A tentative diagnosis of amœbic hepatitis and abscess had been made. X ray examination of the diaphragm had revealed no deformity.

The patient had then been given emetine in doses of 0·18 gramme (three grains) of bismuth emetine iodide for twelve days. The effect had been miraculous. The patient, from being a very sick man, had become well in a week, the jaundice had disappeared and the liver reduced in size. No amœbæ had been found in the stools, but these organisms were very difficult to find. A sigmoidoscopic examination showed no sign of ulceration. The diagnosis of amœbic hepatitis had been made by the process of exclusion and as a result of the response to treatment. An interesting fact was that the patient had never lived away from the north coast area of New South Wales and the south coast area of Queensland.

Aortic Regurgitation and Intestinal Hæmorrhage.

For Dr. Ellis Murphy Dr. Carrol showed a male patient, aged forty years, who had been in the hospital previously two or three times with attacks of dyspnœa and palpitation. The patient suffered definitely from aortic regurgitation.

This time he had been admitted with dyspnœa which had not settled down as previously in seven or ten days. A few nights after admission he had complained of pain in the abdomen and next morning 150 to 180 cubic centimetres (five to six ounces) of dark blood had been passed *per rectum*. Later in the same morning a large quantity of bright blood had been passed, with no symptoms. The patient had been very shocked, the pulse rate had been raised and the colour poor, and he had only gradually picked up. He had then developed signs of fluid in the left side of the chest which had been needled four times, about 900 cubic centimetres (thirty ounces) of fluid being removed. This fluid had once been blood-stained. The temperature had been raised to 37·2° and 37·8° C. (99° and 100° F.). The spleen was just palpable. No growth had followed an attempt at blood culture. A blood count had been done with the following results:

Red cells, per cubic millimetre	2,700,000
White cells, per cubic millimetre	12,000
Hæmoglobin value	50%

Anisocytosis and polychromasia were present. The platelets were normal.

Dr. Murphy wished the opinion of the meeting on the patient. He wanted to know whether the intestinal hæmorrhage and the blood-stained pleural fluid were associated with the aortic regurgitation or not.

Australasian Medical Publishing Company, Limited.

ANNUAL MEETING.

The annual meeting of the Australasian Medical Publishing Company, Limited, was held at The Printing House, Seamer Street, Glebe, on October 1, 1930.

Directors' Report.

The Directors submit their report for the past year and the balance sheet as at June 30, 1930, together with the profit and loss account for the twelve months ended June 30, 1930.

By the sad death of Dr. H. W. Armit considerable readjustment of arrangements for carrying on the work of The Medical Journal of Australia and The Printing House was necessitated. That it was brought about without any break in continuity either in the production of the journal or in the Company's printing business was due to the loyalty and energy of Dr. M. Archdall who took immediate full charge of the editorial department, and of Mr. A. Simpson and the business and printing staffs under him, who adapted themselves at once to the altered circumstances.

Despite the extraordinary economic circumstances existing in the Commonwealth, accentuated as they have been by similar conditions elsewhere, apparently of a world-wide nature, the business of the Company has been successfully carried on.

Like other trading organizations, however, the Company has shared and is continuing to share, the effects of the financial depression. To meet this definite economies have been effected where it has been possible to do so without impairing the standard of the Company's publications or the quality of its printing work.

The Medical Journal of Australia has maintained its position among the scientific publications of the world, whilst its usefulness to the Branches and its popularity with members have continued to increase.

It will be gratifying to note that the bank (1923, £5,000) overdraft has been materially reduced at a rate which will bring about its extinction in the course of the next three years.

Steps have been taken to liquidate all the commitments referred to by Mr. Hadley in his report of October 19, 1928, some of them in advance of the date on which they were due to be paid. One result of the year's work is that the Directors have been enabled to authorize the distribution of one year's interest, amounting to £1,997, on the debentures of the Company.

Dr. Gregory Sprott and Dr. D. D. Paton retire from office by rotation in accordance with the Articles of Association (Article 39). They are eligible and present themselves for reelection.

(*Signed*) T. W. Lipscomb,
Chairman.

Election of Directors.

Dr. Gregory Sprott and Dr. D. D. Paton were reelected to the Board of Directors.

Obituary.

CLAUDE SECCOMBE BROWNE.

The news of the death of Dr. Claude Seccombe Browne, which was recorded in these pages on September 20, 1930, came as a shock to his many friends, both in the medical profession and outside it.

Claude Seccombe Browne was born at Singleton, New South Wales, on February 2, 1880. He was the son of the late Arthur David Browne, grazier, and was educated at Sydney Grammar School. His record of achievement both in scholarship and sport was the pride and envy of many of his contemporaries and has been equalled by few since then. At the Junior Public Examination in 1895 he won the University Medal for general proficiency and the medal for French. At the Senior Public Examination he was bracketed equal with another for the John West and the Graham Medals and the University Medals for Latin and Greek. At the matriculation examination the following year he was bracketed equal with the same fellow student for the Cooper Scholarship Number 2 for classics and the Bowman Cameron Scholarship for general proficiency. He also secured first class honours in Latin, French, Greek and mathematics. In his own school he won the Morehead Scholarship and the Senior Knox prize. He was senior prefect and captain of the school. In the world of sport he was equally successful. He captained the school's cricket and football teams and was captain of the boats. He was also a member of the Combined Great Public Schools team. He represented his school in athletics.

It was to be expected that a boy with such a record would do well in any profession that he might choose. With his classical record it is a little surprising that he did not study arts and devote himself to letters. He turned his attention to medicine, however, and was successful. He went into residence at Saint Andrew's College within the University of Sydney and finally graduated Bachelor of Medicine and Master of Surgery with second class honours. At the university he won his blue for both cricket and football and became one of the foundation members of the University Club.

After graduation Browne became resident medical officer at the Royal Prince Alfred Hospital. He then went into practice at Tumut, New South Wales, where he remained until his death. At the outbreak of war he offered his services. He was for two years with the First Australian General Hospital at Rouen and afterwards spent some time in the Australian unit at Harefield.

Claude Seccombe Browne chose to settle in the country. He had a passion for gardening, and his roses, daffodils and hyacinths were one of the sights for those going to Mount Kosciusko. City life held no attractions for him. He would have succeeded anywhere. Those who knew him in his early days, sometimes wondered at his choice. That he chose wisely is reflected in the place that he held in the affection of those among whom he worked, and the record for faithful work that he has left behind

him, a record worthy of the best traditions of general practice. The sympathy of the medical profession is extended to his wife and daughters, his sister and mother.

Dr. T. B. Clouston writes:

As a friend of his schoolboy days, undergraduates together and partners for the past eighteen years in a country practice, I feel that I should place on record my opinion of "Tom" Browne.

As a medical man his ability was unquestioned and I feel sure he would have held his own wherever he elected to practice. Fortunately for this district Macquarie Street had no attractions for him and many owe their lives to his skill and ability.

Not only in medicine will he be missed. His advice was eagerly sought and readily given on all kinds of subjects—finance, farming, stock and sport. His opinion was always sound. He will be missed by all classes and grades of society—this is the finest memorial anyone can earn. Personally, I feel I have lost my brother.

GEORGE WOODS.

We regret to announce the death of Dr. George Woods, which occurred at Geelong, Victoria, on August 19, 1930.

George Woods was born in Parsonstown, Ireland, seventy-two years ago. He was the son of the late William Woods, of "High Park." He studied medicine in his native country and secured the diploma of Licentiate of the King and Queen's College of Physicians and Surgeons of Ireland. He also became a Licentiate in Midwifery. After graduation he came to Australia and practised in Clarendon, South Australia, for sixteen years. He visited Ireland and remained there for two years. After this time at home he returned to Australia and resumed practice in South Australia, this time at Millicent. After six years there followed another trip to Ireland and on his return to Australia he settled in Geelong, where he remained until his death.

George Woods lived a quiet, unobtrusive life. He was fond of sport; he was a good shot and won many trophies; he was a good tennis player and was very fond of golf. He loved his professional work and took a keen interest in it until a few weeks before his death. He cared little for social life, but preferred the pleasures of home and the company of the members of his family. To them the sympathy of a large circle of friends has been extended.

Special Correspondence.

LONDON LETTER.

By Our Special Correspondent.

Medical Museums.

Medical museums are now regarded as a necessary adjunct to post-graduate instruction and every teaching hospital has its own museum, consisting of specimens obtained from the hospital itself. It is gradually being realized, however, that museums intended primarily for the training of students are not necessarily suitable for post-graduates, and also that properly constructed medical museums may be of definite propaganda value to the lay public. The advance of science in every form has contributed greatly to the expansion and increased usefulness of the modern medical museum. In addition to specimens illustrating the various diseases, radiographs, electrocardiographs, kinematographs, photographs and paintings contribute their quota of knowledge, and owing to the invention of new instruments for seeing inside the body exact pictures can be made. The Royal College of Physicians of London possesses a museum, as does the Royal College of Surgeons of England. The annual report of the latter (by Sir Arthur Keith, Conservator of the Museum) has just been published and makes interesting reading. The space hitherto devoted to the museum at the Royal College of Surgeons has proved far too small and has not permitted of adequate accommodation being spared to research students. This fault has now been remedied and the report contains a plan showing the newly added laboratories and store rooms. Owing to the activity of the President of the College (Lord Moynihan) three scholarships will shortly be available—the Beaverbrook, the Melchett, and the Bernhard Baron—each yielding £500 *per annum*. The existing catalogues of the various departments (pathological, physiological, human osteology) and collections (teratological, laryngological, odontological, and the historical section) have been revised, brought up to date and in some instances are being reedited, and important additions and donations have been made. In particular, the cataloguing and arrangement has been completed of the "Strangeways Collection" which consists of specimens illustrative of various lesions of chronic arthritis and of certain disorders of bone. The report concludes with a list of the receptions given by the College during the year to various associations and of the lectures and demonstrations given within its walls.

An independent museum, that is to say, a museum which is not attached to any college or hospital, is the Wellcome Museum of Medical Science, 25, Gordon Street, W.C.1. Dr. S. H. Daukes, the Director, has recently published a book setting forth the aims and objects of the ideal medical museum, which should be designed not only for members of the medical profession, but should appeal also to the lay public. Unlike other museums, a medical museum should not be only a record of past efforts, but should be kept up to date, and should in particular emphasize the necessity of looking to the future instead of resting content upon the successes of the past. As medicine is becoming more and more specialized, it is increasingly necessary to have museums where can be studied all phases of special diseases and in particular their relation to medicine as a whole. It should be a *sine qua non* that museum study forms a definite part of teaching, whether undergraduate or post-graduate. A properly equipped museum can fill several functions: the collection of specimens *et cetera* illustrating disease for reference and for comparison, the formation of a centre for research work, the provision of facilities for revision and post-graduate work.

Museums may be divided into five classes: Those attached to teaching hospitals (already previously mentioned); national (the Wellcome Museum of Medical Science is an example), which should include the education of the lay public among its functions; special, which, as a general rule, are solely used by specialists; private: public.

The cataloguing of the museum must be efficient in regard to its completeness and at the same time must be easy of comprehension; and descriptive labels should be clearly written and affixed to every exhibit in order to save both time and fatigue.

Dr. Daukes states that the system in force at the Wellcome Museum is a result of experiments made at the time when intensive training at the School of Army Hygiene during the war was necessary. It was then found that the best results were obtained when the teaching was given in demonstration centres which as nearly as possible reproduced front line conditions. This principle can and should be carried out in any teaching of medical subjects, when it will be found that a properly conducted museum demonstration is of more value than a lecture.

As regards the general public, museum demonstrations have been organized by Dr. Daukes at the request of the Ministry of Health, not only at the Wembley Exhibition in 1924 (on "Tropical Diseases") and also in 1925 (on "Home Hygiene and Disease Prevention"), but also at the Antwerp Exhibition at present in progress.

Much depends upon the arrangement of the medical museum and the care with which the sections are planned. Photographs, paintings, cultures, wax models, actual specimens, all should be pressed into service, and the section should be so arranged that it deals not only with the particular disease, but also with its relation to medicine as a whole. It is obvious that there must be close cooperation between the pathological department, the clinical staff and the museum itself.

Correspondence.

MEDICAL ARTICLES IN THE DAILY PRESS.

SIR: In Saturday's issue last of one of the most widely circulated dailies of Sydney appeared a report concerning the work of one of Sydney's surgeons, the name of the surgeon being published.

I consider that By-Law 10*a* of the Memorandum and Articles of Association and By-Laws of the New South Wales Branch of the Association indicates unmistakably the attitude to be adopted by members in the conduct of their practice. I object to the article in question appearing as it did and consider that if the surgeon concerned does not take steps to repudiate any connexion with the insertion of the article, the Council of the Branch should at once, through the columns of the press which offended, make clear its attitude.

Yours, etc.,

F. TIPPING.

Bogan Gate,
New South Wales.
September 16, 1930.

[The attention of Dr. Tipping is drawn to a letter from Dr. T. W. Lipscomb, Chairman of Directors, Australasian Medical Publishing Company, Limited, published in the issue of September 27, 1930, at page 457.—EDITOR.]

MODERN VIEWS ON SOME OBSTETRIC AND GYNÆCOLOGICAL PROBLEMS.

SIR: In THE MEDICAL JOURNAL OF AUSTRALIA of September 6 Dr. Ralph Worrall replied to letters written by Dr. Robert Fowler and myself. In this reply he has gone to great lengths to prove that surgery is far superior to radium in the treatment of numerous non-malignant conditions. Dr. Fowler in his letter spoke only of radium in its treatment of malignant conditions (carcinoma of the *cervix uteri*), and nine-tenths of my letter was concerned with the same subject. I concluded by making a passing reference to the treatment of non-malignant conditions. However, while on this subject I notice that Dr. Worrall has named thirteen "after effects" from radium, varying between "burn of the vagina" and pelvic peritonitis.

Any radiologist will admit that practically every one of these "after-effects" can be avoided if the worker knows the properties and dangers of the element he is using. There is no doubt that the burn of the vagina was the result of using insufficiently filtered radium, while pyometra, pelvic abscess and pelvic peritonitis should never occur if the operator is careful enough in his examination of the pelvis. If there is any inflammatory condition present in the adnexa, radium is absolutely contra-indicated, and if it is used before any such condition has subsided, any of these three complications mentioned will arise. In these cases the operator is to blame and not the radium which he is using.

Dr. Worrall admits that Heyman, of Stockholm, has achieved results equal to those of surgery, but claims that no British surgeon, using the same technique, has obtained similar results. British surgeons have not obtained the results Heyman has because they have not followed him to the letter. Heyman himself complained to me that British surgeons were professing to use his technique, but were in reality using their own modification of it—and this is very true. Further, Dr. Worrall must remember that it is only in the last three or four years that anything like adequate supplies of radium have been available in England and before this there was a natural tendency to try and treat the maximum number with the small amount of radium element at their disposal. In England the work is done in hospitals and not in clinics, as in Stockholm. Again, the seemingly poorer results in England are due in no small measure to a poor "follow-up" (every case lost sight of is counted as having died of the disease), whereas in Sweden, Heyman is in the happy position of being able to trace every case he has treated. This is made possible by the cooperation of the Swedish Government which grants free railway tickets to patients visiting Radiumhemmet for treatment or "follow-up." Now, however, matters have improved a great deal in England and I am sure that the future will show that British surgeons can and do obtain the same results as Continental workers, especially as they are using "intra-peritoneal" radium energetically.

Dr. Worrall states that even Heyman cannot show the ten and twenty year survivals which he and other foremost surgeons can. This is not true. Even in my short stay at Radiumhemmet I saw several, and their statistics show that four cases treated in 1914, nine cases in 1915, one in 1916 and seven in 1917 were alive and well ten years later.

Yours, etc.,

HUBERT K. PORTER.

"Clorinda,"
Darling Street,
Balmain.
September 9, 1930.

PUERPERAL HÆMORRHAGE.

SIR: In Dr. E. Sydney Morris's excellent paper published in THE MEDICAL JOURNAL OF AUSTRALIA on September 6, 1930, he remarks:

> Should *post partum* hæmorrhage be controlled by packing of the uterus or is this unphysiological, since the uterus cannot be kept contracted and retracted with gauze inside it? I realize that it may be a last effort to control a desperate situation, but the evidence so far does not fill one with enthusiasm for its effectiveness.

In the treatment of *post partum* hæmorrhage Herman's "Difficult Labour" (1920) states:

> Plugging the uterus with iodoform gauze—this method was recommended by Duhrssen. It is neither certain nor safe and is seldom used in this country. Like the injection of a styptic, it is unphysiological, for the uterus cannot be completely contracted while the gauze is inside it.

On the other hand, I turn to Fothergill, of my student days. Here I find "Packing the uterine cavity."

> There are few cases in which hot water fails to check *post partum* hæmorrhage. In these rare cases tightly packing of the uterus with an antiseptic substance exerts pressure on the bleeding surface and stimulates contraction. Iodoform gauze is the best material for packing. Several yards of gauze will be required, as the packing must be very firm and must be pushed right up to the fundus.

De Lee recommends it strongly and states:

> In all but a very few cases packing will stop the bleeding. Chrobak, Ahlfeld, Bumm, von Braun report 10 failures in 120 cases, but an exact study of the histories shows that too much was expected of the operation. The author has plugged the uterus over 100 times for conditions not due to lacerations. Only twice was it necessary to replace the pack with a second one.

He goes on to say:

> Without doubt most of the reported failures are due to poor technique; the gauze is not put in correctly.

From my own experience I have found plugging with gauze most efficacious in controlling hæmorrhage after having to empty the uterus in cases of incomplete abortion and miscarriage. Not only does it stop the hæmorrhage by pressure, but certainly stimulates the uterus to contract and retract and in these cases removal of the gauze twelve to twenty-four hours afterwards from a firmly retracted uterus makes quite a vivid impression on one's

memory. My experience in plugging the uterus in cases of *post partum* hæmorrhage has been equally as happy. The uterus contracts down firmly on the iodoform gauze which should be about three inches wide and six layers in thickness and should be inserted firmly up to the fundus. It is surprising how quickly the packing can be done and how little gauze is required—certainly not as a rule more than a yard. In the discussion that followed Dr. J. A. Cameron's paper on *post partum* hæmorrhage at the meeting of the Australasian Medical Congress, September, 1929, I stated that packing of the uterus was a very efficacious method for arresting hæmorrhage after delivery of the placenta when the usual methods had failed. It was easy to do and did not require a great amount of packing.

In this I was supported by the President of the Section of Obstetrics and Gynæcology (Dr. Jellett) who said that he thought that plugging with gauze was the sheet anchor of the obstetrician in treating patients who did not respond to other methods. He did not find that a large amount of gauze was necessary to plug the uterus.

Yours, etc.,

JAMES HUGHES, M.B., Ch.M. (Sydney).

215, Macquarie Street,
Sydney.
September 11, 1930.

Books Received.

CANCER OF THE LUNG AND OTHER INTRATHORACIC TUMOURS, by Maurice Davidson, M.A., M.D., B.Ch., F.R.C.P., with a foreword by Arthur J. Hall, M.A., M.D., D.Sc., F.R.C.P.; 1930. Bristol: John Wright and Sons Limited. Crown 4to., pp. 184, with illustrations. Price: 17s. 6d. net.

PRINCIPLES AND PRACTICE OF DERMATOLOGY, VOLUME III: THE TREATMENT OF SKIN DISEASES IN DETAIL, by Noxon Toomey, M.D., B.A., F.A.C.P.; 1930. Saint Louis: The Lister Medical Press. Royal 8vo., pp. 512.

Diary for the Month.

OCT. 21.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
OCT. 24.—Queensland Branch, B.M.A.: Council.
OCT. 28.—Queensland Branch, B.M.A.: Obstetrical Section.
OCT. 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
OCT. 30.—New South Wales Branch, B.M.A.: Branch.
OCT. 30.—South Australian Branch, B.M.A.: Branch.

Medical Appointments.

Dr. E. H. Miles (B.M.A.) has been appointed Government Medical Officer at Yeoval, New South Wales.

* * *

Dr. V. A. Conlon has been appointed Government Medical Officer at Lockhart, New South Wales.

* * *

Dr. C. H. Rowe (B.M.A.) has been appointed an Official Visitor of the Mental Diseases Hospital, New Norfolk, Tasmania, until December 31, 1930.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xvi.

AUGATHELLA HOSPITAL, QUEENSLAND: Medical Superintendent.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members desiring to accept appointment in ANY COUNTRY HOSPITAL, are advised to submit a copy of their agreement to the Council before signing, in their own interests. Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Hospital. Mount Isa Mines.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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